THE

VETERINARY JOURNAL

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VIS UNITA FORTIOR.

AT the present time it is most desirable that the veterinary profession should persuade the public of its paramount importance in matters pertaining to the pastoral industry and many branches of public health, and it is of the utmost importance, too, that its expressed opinions should be unanimous; for if its members differ among themselves the public will lose confidence in the profession as a whole.

But, alas, it has to be admitted that there is often a wide divergence of opinion, even in matters of primary importance, not only among the so-called experts, but among general practitioners. For example, opinions differ as to the best means of ensuring pure milk and the efficacy of pasteurisation; the value of the tuberculin test, and the best method of applying it; the best course to pursue in the control of such diseases as contagious abortion, Jöhnes disease, foot-and-mouth disease, swine fever, and many others which constitute a constant menace to the stock industries of Great Britain and her Colonies. Even in such matters as education, recruitment and appointments, considerable differences of opinion exist.

Under existing conditions this state of affairs has been almost inevitable, for it is rarely possible for the members of the profession, few in number but widely scattered, to meet together and discuss matters in such a way as to arrive at a final solution of their many problems, and to agree to a definite line of policy to which all can conscientiously adhere. The only manner in which the considered opinions of our members can be presented, considered and criticised, and some finality and unanimity arrived at, is through the medium of the veterinary press. Even then the discussion is generally of a somewhat desultory nature, for it is rarely possible to devote the whole of any issue of existing publications to the consideration of any one problem.

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For some time past THE VETERINARY JOURNAL has issued special numbers devoted to special subjects, and informative articles have been presented by contributors having special knowledge of the diseases under consideration. But with the presentation of such articles the matter has dropped, and there has been little or no discussion, or criticism, by those equally competent to express an opinion.

It is therefore suggested for the consideration of our readers that in future a subject of primary importance should be presented in all its aspects in one number, and the views presented should be criticised in the next; and that the Editor should finally " sum up," and in an Editorial should express what appears to him to represent the considered opinion of the profession on the particular matter under discussion. In this way it is hoped that the members of the profession will be rendered *au fait* with the recent advances in veterinary science and the views of their *confrères*, and will be able to express with unanimity the considered opinion of the profession which they represent. As a result, many of those conflicting opinions which go to undermine the confidence of the public may be avoided, and the truth of our motto, VIS UNITA FORTIOR, will be demonstrated, not only to our advantage, but also to the advantage of the animals and their owners.

THE CONQUEST OF DISTEMPER.

ON a further page of this issue is published the final portion of the scientific report of the *Field* Distemper Council, and it is a good deed done for the profession that a method has now been scientifically devised whereby this scourge of dogdom has been conquered. Too much praise cannot be given to the workers whose patient research into the vagaries of this ultra-visible virus has brought about this gratifying result, which is now not only a satisfactory one in laboratory animals, but also in the kennel outside; and the names of Laidlaw, Dunkin and Dalling will always be remembered with gratitude by the owners of valuable dogs and the commercial fur-bearing animals. In expressing the profession's gratitude, one must not quite forget the part played by those who, by their perseverance and persistent efforts, obtained the necessary £30,000, without which the investigation could not have been done. THE VETERINARY JOURNAL played its part; in fact it was the Editor who gave the fund its start, and the columns of this journal were opened up for subscriptions at the same time that the appeal started in the *Field*.

The former Editor of the latter paper, the late Sir Theodore Cook, took a great personal interest in the progress of the fund right from the start, as did also one of his colleagues on the staff, the late Dr. Hammond Smith; whilst the tactful methods devised for attracting subscriptions from the public (not only of Great Britain, but also America) adopted by the organising secretary, Mr. Moss-Blundell, were a masterpiece. Between them all the combination was one of the strongest which has ever worked together, and the result has justified the expenditure.

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General Articles

THE PREVENTION OF DOG DISTEMPER: FINAL REPORT OF THE "FIELD" DISTEMPER COUNCIL.

THE researches promoted by the *Field* Distemper Council into the cause, prevention and treatment of canine distemper, have been successfully concluded. The results achieved may therefore be briefly reviewed, in supplement to the more detailed progress reports already published, and a final expression of gratitude made for all the help so generously given to the work by British and American subscribers. When the Fund was inaugurated ten years ago, distemper was regarded as of almost inevitable occurrence in every dog's life, and caused either death or serious after-effects in a large proportion of cases. No trustworthy means for its prevention was known; the only treatment was purely empirical and palliative; "distemper" was not always clearly distinguishable from other diseases, and the very nature of the causative organism was in dispute.

The position has now been completely changed as the result of the work done by Dr. P. P. Laidlaw, F.R.S., and Mr. G. W. Dunkin, M.R.C.V.S., with the resources placed at their disposal from the *Field* Distemper Fund, and by the Medical Research Council. The cause of the disease has been clearly defined, and its nature is better understood. Two methods of protective inoculation have been devised, and trial on a large scale has shown them to be successful. A useful method of curative treatment has also been introduced. The subsequent work of commercial laboratories in Great Britain and the United States has now made these results available to the veterinary profession, and so to the public for the benefit of dogs of every kind. The principal steps that have led to this great change may be briefly recapitulated: (1) Proof was early obtained that primary distemper is due to an ultra-microscopic filter-passing virus. This knowledge provided a rational basis for new means of prevention and treatment. (2) It was shown that distemper can be readily transmitted from ferret to dog and *vice versa*, and this fact was of the greatest assistance in the subsequent experimental work. Other studies were made of the infectivity, symptoms, and pathology of primary distemper. (3) Eventually it was found possible to produce the following: *Virus* containing the living organisms of the disease, and capable of severely infecting a susceptible animal. *Vaccine* containing the killed organisms of the disease. *Anti-serum* from the blood of an animal that has itself been made strongly immune by repeated administrations of virus. (4) It was found that a healthy dog could be given lasting protection against distemper infection by the inoculation of vaccine followed by inoculation of virus a fortnight later. (5) It was later found that an animal could also be immunised by the simultaneous inoculation, on different sides of the body, of anti-serum and virus.

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(6) It was found that the anti-serum, used alone, was of value in lessening the severity of an attack of distemper when given early enough in the disease ; it is also useful for giving temporary protection to animals that have recently been exposed to infection. (7) The experimental use of these different methods was extended to trials on a large scale with dogs under ordinary conditions, notably kennels of fox-hounds. Highly successful results were obtained. A subsequent survey of the results with the vaccine-virus method showed that, in cases where exposure to infection was certain, the incidence of distemper among 650 hounds belonging to twenty-three hunting packs was only 1.4 per cent., and the death-rate from this cause 0.3 per cent. Without inoculation, the incidence among young fox-hounds in this country is nearly 100 per cent., and the death-rate is frequently 50 per cent., and may exceed 75 per cent. (8) With a view to facilitating manufacture for general use, many highly technical questions as to the mode of preparation, standardisation, and stability of the different products were investigated and solved. The research having thus been completely successful, as shown not only by experiment, but also by large-scale trials under ordinary conditions, the supply of the products passed to commercial laboratories in Great Britain and in the United States. The early results of this were not uniformly satisfactory in this country, owing in part to special difficulties at first found by the manufacturers in rapidly setting up large-scale production to meet an enormous and urgent demand from dog lovers, and in part to the novelty of the method, and to some want of appreciation of the proper conditions for its use. More recent experience of the general supply shows that the vaccine-virus method is now successful in a very high percentage of cases. Experience of serum-virus immunisation is as yet less adequate, but the method involves only a single attendance by the veterinary practitioner, and is often preferred by reason of its practical convenience. The use of serum alone for curative treatment is giving good results. These achievements may soon have a wider interest. True distemper has recently been found to occur among fur-bearing animals related to the dog (silver fox) or ferret (fitch, mink, fisher), and preliminary experiments already show that the methods now available will have immediate value where these animals are farmed. The work primarily undertaken for the protection of dogs seems likely, therefore, to save an important industry from losses which have hitherto been often disastrous. Apart from all else, the knowledge gained of this virus disease and of the means of combating it cannot fail to have a far-reaching and helpful influence upon the progress of research into virus diseases generally, whether of animals or of man himself ; there is evidence of this already. Thus we have gained, as the result of the research work, and of the subsequent enterprise of the commercial laboratories, a completely successful method of protecting dogs and other animals against distemper. There is an alternative method which has some practical advantages, but of which the value is not, at least for the present, so fully established. We have also a useful method for the curative treatment of the disease in its early stages.

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In conclusion, the *Field* Distemper Council desire to express their very high appreciation of the brilliant manner in which the investigators, Dr. Laidlaw and Mr. Dunkin, have brought their researches, with great labour and by the use of new methods, to so successful an issue in a field of great difficulty. Second only to this in importance has been the generous provision of material resources by the British and American public in voluntary donations, and by Parliament here through the Medical Research Council. These resources, and the allowance of ample time, have enabled the work to be done with thoroughness, and with every precaution against accident or fallacy, and step by step, with unhurried care.

DISEASES OF AVIARY BIRDS.

By A. E. HAMERTON, M.R.C.S.
Pathologist to the Zoological Society of London.

DISEASES causing mortality among birds that are commonly kept in captivity may be specified in order of frequency as follows :

(1) *Diseases of the Respiratory System*, including (*a*) congestion and œdema of lungs ; (*b*) pneumonia ; (*c*) bronchitis and bronchopneumonia. In the aviaries of the London Zoological Gardens, where my experience has been gained, the respiratory diseases above named are by far the most destructive, and affect mainly small species. The lungs of birds are so closely fixed to the parietes that there is little room for expansion ; any inflammatory condition is therefore often quickly fatal. Congestion of the lungs may be defined as a condition of active hyperæmia of the organs, that is revealed by areas of deep-red, almost purple, discoloration, which on compression between finger and thumb exude frothy fluid. The portion of lung affected contains some air, and does not sink in water. The whole lung is œdematous and boggy from oozing of fluid into the air spaces. On microscopic examination the air spaces of the lung are patent, but most of them contain some coagulum, leucocytes, and red blood cells. An extension of this process results in plugging of the tertiary bronchi and their diverticula with exudate, and consolidation of groups of lobules, which form a patch of consolidated lung that sinks in water, a condition of bronchopneumonia. Sometimes the whole of one lung and a large part of the other may be found firmly consolidated and hepatized, and sinks like a stone in water ; a condition resembling acute lobar pneumonia of mammals. These conditions come on suddenly : the bird may appear well in the evening, and is found dead at the bottom of its cage next morning. The disease is not usually diagnosed before death, and I know of no treatment. Cold and fog, or a dusty stuffy atmosphere appear to be predisposing causes.

(2) *Diseases of the Alimentary System.*—(*a*) Catarrh of intestine ; (*b*) enteritis and gastritis ; (*c*) fæcal impaction and intestinal obstruction. Catarrh of the intestines and enteritis are the next most common causes of death in cage birds. In most cases no specific bacterial or parasitic cause can be determined, and the

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distinction between catarrhal inflammation and enteritis is somewhat arbitrary, and depends mainly upon the intensity of the lesions as seen by the naked eye, or as may be indicated before death by the character of the diarrhœa from which the birds suffer. In catarrh of the intestines there is swelling and œdema of the intestinal mucosa, and in chronic cases the mucosa may have atrophied and disappeared from portions of the bowel. The lumen of the gut often contains undigested seeds or sand, and there is always great excess of mucous exudate. In enteritis the gut is more severely and more extensively inflamed, and deep red in colour ; the mucosa is intensely congested and hæmorrhagic, with patches of denudation, sloughing or ulceration, and a blood-stained exudate, or even blood may fill the lumen of the gut. Both conditions are frequently associated with secondary inflammation of the pancreas and gall bladder, viscosity of bile, and changes in the liver associated with biliary stasis.

Local ulceration of the gizzard and intestine are not uncommon, and are frequently caused by the irritation of a foreign body, usually a piece of wire, which may perforate the walls of the alimentary canal and cause fatal peritonitis. Idiopathic peptic ulcers occasionally occur in the gizzard, but more commonly in the duodenum near the entrance of the pancreatic ducts, and may cause death by perforation, hæmorrhage, or secondary septic processes in pancreas or liver. Intestinal obstruction and ulceration caused by impaction of chalk-like concretions of excreta (urates, etc.) in the cloaca, and secondary fatal septicæmia is a condition to which birds in captivity are liable. In small birds much the same effect may be produced externally by stoppage of the vent, due to the accumulation of hardened fæcal material on the surrounding feathers.

Little can be done in London in the way of preventing the respiratory diseases, for they are climatic in origin, and associated with the presence of fog, atmospheric impurities, cold weather, and stuffy, dusty rooms. Much, however, can be done in reducing the mortality from intestinal diseases, for they, like mycosis and tuberculosis, are to a great extent filth diseases engendered in cages with dirty perches, sides, and bottoms, dirty feeding utensils, and feeding with stale food. Wholesome, fresh food, cleanliness, and the use of disinfectants are the remedies. Troubles arising from constipation and over-feeding can sometimes be recognised during life, and successfully treated with household remedies.

(3) *Specific Infections*, the cause of which is definitely known, and can easily be determined by simple laboratory methods, viz.—Tuberculosis (avian) ; and mycosis. It is of interest to recall that tuberculosis of birds was first recognised by Bland Sutton when working in the Prosectorium of the London Zoological Gardens in 1884. His material was examined by Drs. Gibbs and Klein, then the accepted authorities on tubercle bacilli in London. At first the disease was regarded as leprosy ; later Klein changed his opinion, and agreed that the disease was tuberculous.[1] Cage birds of all kinds are susceptible to the avian type of tubercle bacillus ; but with the exception of cockatoos and parrots, they are said to be resistant to the human and bovine strains. The

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only mammal of economic importance that is commonly infected with avian tubercle bacilli is the domestic pig, and it is possible that in the very rare cases in which man has become infected with the avian type of tubercle bacillus he has contracted it through the pig. Calves are susceptible to the avian type of tubercle bacillus, but to a much less degree. In the Zoological Gardens a kangaroo was recently found infected with avian tubercle bacilli in the pancreas, and Stanley Griffith has reported a similar infection of the udder of a goat.[2]

Although tuberculosis probably attacks every avian species living in captivity or domestication, it seems to be extremely rare among wild birds. The authenticated cases on record are so few that they may be mentioned. Shattock and others described a case in a lapwing (*Vanellus vanellus*) which was captured in Scotland. The specimen is now in the Museum of the College of Surgeons. Hammond Smith records a case in a wild kestrel (*Falco tinnunculus*). Cobbett found a case in a partridge (*Perdix perdix*), and Hare, of the Royal Veterinary College, quite recently described a case in a wild blackbird (*Turdus merula*) caught by a cat in a London garden.[3]

In most birds affected with tuberculosis the lesions are large, and conspicuous by advanced caseation. The liver, spleen and intestines are in most cases the organs most severely attacked. Sometimes the disease may be confined to the respiratory system, in which case the trachea, lungs and air sacs may be affected. In these cases it is often impossible to distinguish the lesion from mycosis, except by microscopical examination, when the finding of tubercle bacilli is decisive. I have never found mycotic lesions and tuberculous lesions co-existing. In records of such double infections I can find no data regarding microscopical examination, which in certain cases is the only means of distinguishing between the two diseases. Tuberculosis of the avian glands (thymus or lymph glands of neck) is fairly common, and in fact there is no internal avian tissue or structure, except that of the nervous system, in which I have not found lesions of tuberculosis. If an infected bird is introduced, the aviary becomes grossly contaminated, and other cases may follow, for the birds pick up the tubercle bacilli in millions off the ground with their food, etc.

The following case is illustrative: A Crossoptilon pheasant (*Crossoptilon auritum*) died from " enteritis." Examination with a hand lens revealed minute white deposits scattered throughout the whole extent of the intestinal mucosa. Smear preparations of the fæces were examined microscopically. They resembled preparations made from a pure culture of tubercle bacilli in the vast numbers of these organisms revealed. Histological examination of the tissue showed the deposits to be miliary tubercles in the mucosa and intestinal villi, which were packed with tubercle bacilli. The potential infectivity of this bird needs no emphasis.

Another case illustrates localised quiescent tuberculosis: A Jackson's Whydah bird (*Drepanoplectes jacksoni*) lived in the Zoo for twenty-one years, when it died from the pressure effects of an abdominal " tumour," the size of a filbert nut, that occupied the greater part of the abdomen. The tumour was

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enclosed in a fibrous capsule, and contained caseous material. Microscopical examination revealed masses of tubercle bacilli in a structureless matrix, with giant-cell systems in the enveloping capsule. The " tumour " was found to be the spleen ; its proper tissue was destroyed. No other tuberculous deposits were found in the bird. Despite the bird's age and destruction of its spleen, its general condition was good. It was in full nuptial plumage, and the testicles showed normal seasonal activity. Interesting points in this case are : (1) The partiality of the avian tubercle bacillus for the spleen of birds in contrast to the rarity of tuberculosis of the human spleen ; (2) Birds, like mankind, can get on very well without any spleen at all.

Mycosis is a common and fatal disease of the air passages due to infection with fungi (moulds) of the genera aspergillus and penicillium. It is characterised by the formation of a mouldy growth on the mucous membranes of the air tubes, and of the air sacs in the thoracic and abdominal cavities. The disease occurs mainly among exotic birds, and is probably acquired under the insanitary conditions in dealers' stores and on the voyage to England. We have seen whole consignments of birds arrive, crowded together in cages fouled with mouldy excreta, in which nearly every bird has died from mycosis shortly after arrival. Aspergillus fumigatus is the commonest fungus causing mycotic lesions in birds. The primary deposit usually occurs on the inner surface of the large thoracic and abdominal air sacs, in which they spread and coalesce, to form a tough, yellowish, leathery, mouldy film, lining the whole of the inner surface of large air sacs ; or the growth may completely fill the air sacs of small birds with hard yellow nodules, which may occur in the air spaces of the lungs, where they cause inflammatory consolidation, distinguishable from tuberculosis only by microscopical examination (Figs. 1 and 2). In some cases the mycotic growth is found only in the syrinx, where it causes death by choking of the windpipe (Fig. 3). The disease kills slowly, and can often be diagnosed during life by the gasping and laboured respiration of the birds. When recognised, a sick bird should be segregated.

(4) *Deaths Caused by Animal Parasites.*—External parasites are responsible for ill-health among aviary and domesticated birds that are kept under insanitary conditions, but they are rarely a primary cause of death. More than forty species of lice occur among aviary birds. The common bird flea (*Pulex avium*) infests many species of birds, and the red mite is a nocturnal blood sucker that causes loss of blood and intense irritation. Scaly leg or foot mange is caused by a species of sarcoptic mite, and another species infests the portions of the quills that lie beneath the skin, and causes loss of feathers. The fowl tick infects poultry and canaries, and carries the fatal spirillum fever of birds—fortunately a rare disease in England. Internal parasites may be dangerous to birds, and often cause death.

The following are common and destructive among aviary birds :—

Gape Worms (*Syngamus trachealus*) infest the air passages of certain cage birds, waterfowl and pheasants, and cause death by blocking of the air passages, or by causing inflammatory conditions of the lungs.

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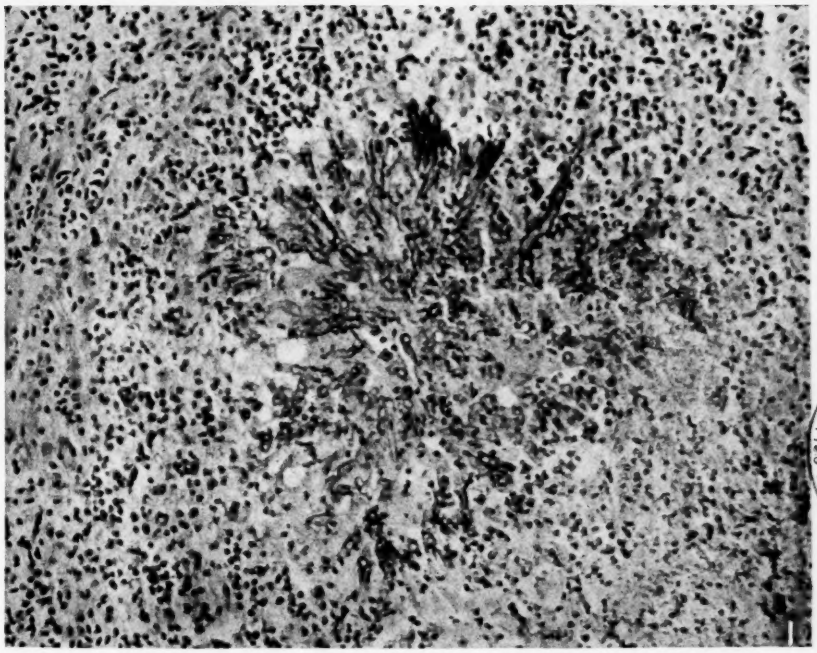

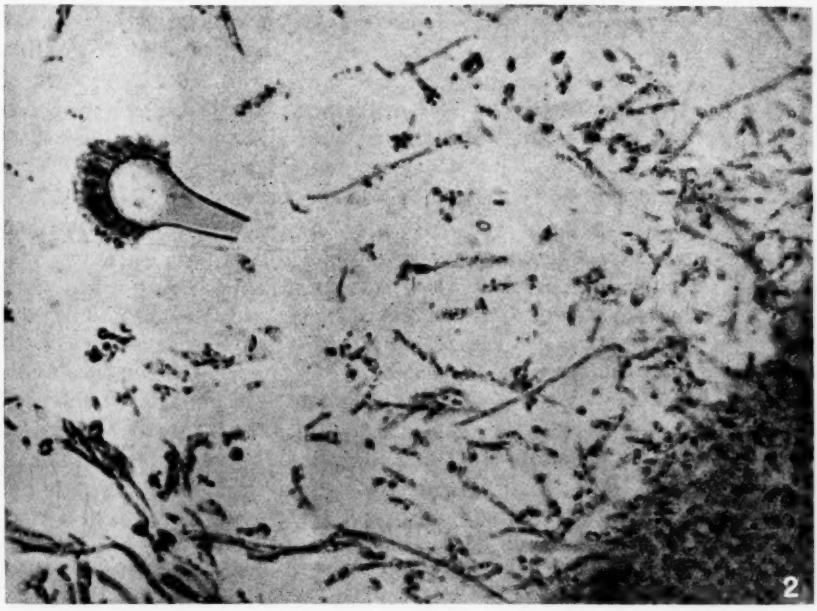

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PHOTOMICROGRAPHS OF TYPICAL LESIONS IN AVIAN PULMONARY MYCOSIS.
(By the courtesy of the Zoological Society, London.)

FIG. 1. Nodule of *Aspergillus fumigatus* in lung of a penguin. From the periphery of the nodule, hyphæ radiate out towards the air vesicles, within which fruit-heads (conidophores) (FIG. 2) may be seen discharging spores into the air spaces.

(Article by A. E. Hamerton, M.R.C.S., *page* 5)

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Round Worms (*Nematodes*) are common intestinal parasites, and sometimes cause death by perforating the gut. The most destructive, however, are the following :—

Heterakis.—Small round worms from 31–2 cm. in length, easily seen by the naked eye, infest the cæca of birds, and cause severe inflammation and gangrene of this part of the bowel. It is a most destructive disease amongst gallinaceous birds and pigeons.

Spiroptera, or hair worms, infest the gizzard and proventiculous of waterfowl and gallinaceous birds, and cause death by burrowing under the mucosa and setting up severe inflammation.

A *Filaria*-like worm (*Diplotriæna tricuspis*) is sometimes found in the body cavity of jays that have died of pneumonic consolidation of the lungs. This worm causes fatal avian pneumonia by laying its eggs in vast numbers in the tissues of the lung, in which they can be readily detected on microscopical examination of the affected organ.

Tapeworms (*Cestodes*) are common intestinal parasites of aviary birds. They do not usually cause disease, except when found in very large numbers ; they may cause death by intestinal obstruction or inflammation.

Flukes or *Flatworms* (*Trematodes*) are sometimes found infesting the kidneys of egrets, and possibly other water birds. If in large numbers, they may set up a verminous nephritis, resulting in death from disorganisation of the kidneys. The eggs of the parasite are readily found on microscopical examination of the contents of ureters or cloaca.

Microfilaria.—The larval forms of filaria worms are sometimes found concentrated in great numbers in the capillaries and alveoli of the lungs of birds that have died from pulmonary congestion and œdema, and can hardly be acquitted of being concerned in causing the fatal lesion.

The air-sac mite (*Cytolechus nuedus*) infests the air sacs, and may cause purulent inflammation therein.

Coccidia are protozoal parasites infesting the intestinal epithelium of birds. They set up intractable diarrhœa that is most destructive to young birds. Coccidiosis can be diagnosed only by microscopical examination of the excreta, in which the characteristic oöcyst can easily be recognised. When the parasites infest the cæcal epithelium the lesions produced may closely resemble those caused by heterakis worms ; minute inspection with the naked eye or hand lens will exclude the worm infection.

Other protozoal parasites of birds, *viz.*, those of bird malaria (*Plasmodium*) are of special interest in that they were studied by Ronald Ross, who demonstrated their development in the mosquito, a revelation that led directly to the discovery of the *anophales* mosquito as the essential insect host of the parasite (*plasmodium*) of human malaria[4]. Avian plasmodia infest the red-blood corpuscles of their vertebrate hosts, and produce malarial fever, which is a fairly common cause of death in recently imported tropical birds.

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Newly imported birds will sometimes suddenly drop off their perches dead. Post-mortem examination may show nothing visible to the naked eye: a microscopical examination of the blood, however, will reveal great destruction of red blood cells, more than half of which may be occupied by a malarial parasite.

Diseases of the Circulatory System.—Idiopathic anæmia of birds can rarely be traced to the blood-forming organs. Leukæmia, however, is a fairly common cause of avian mortality, and is manifested by an enormous increase of the leucocytes and decrease in the number of red cells; associated with pale, watery appearance of the blood, and a pallid anæmic aspect of all the internal organs. Pericarditis, with serous or purulent effusion, or a dry form of the disease with a white film or urate deposit on the opposing surfaces of the sac, is commonly found as a secondary complication of grave primary lesions of the kidneys, or in general septicæmia. Birds will sometimes die, however, with the pericardium distended with clear fluid, and a like effusion in the peritoneum, and no other lesion. There is generally a history of exposure in such cases. Myocarditis and fatty degeneration of the heart in old birds is often associated with rupture of the auricles, hæmopericardium and sudden death. Endocarditis, or valvular disease of the heart, is quite a common cause of death, and is generally acute, and often associated with an external septic lesion, such as septic "corns" of feet, or infection of the wound after pinioning, or other injury. Chronic endocarditis resulting in sclerosis and distortion of the mitral valves with incompetence and cardiac hypertrophy occurs in old birds. Fatty and calcareous atheromatous deposits in the arterial system are sometimes so advanced as to seriously diminish the flow of blood by narrowing of the calibre of the brachio-cephalic and coronary arteries, a common disease in aged parrots.

Diseases of the Urinary and Genital Systems.—Birds of all kinds are very subject to nephritis, which is a common cause of death. In nearly all cases of nephritis in captive birds the inflammatory conditions found appear to have arisen from defects in elimination resulting in "renal constipation," *i.e.*, clogging of the urinary tubules and ureters with semi-solid or solid white concretions of urates. In such cases the kidneys are usually enlarged, dirty-yellow in colour, and may show hæmorrhages in the soft, friable and degenerated parenchyma. The condition is often associated with œdema of the lungs and anæmia, pyelonephrosis and septicæmia, and the serous membranes of the heart and abdominal viscera may appear to be white-washed with a film of urates. Apart from the condition above noted, degeneration changes, hyaline, fatty and lipoid, are conspicuous lesions, and affect mainly the tubular epithelium; or the tubules may form cysts devoid of epithelium. There is usually much inflammatory reaction and proliferation of fibrous tissue in the inter-tubular spaces, and round the glomeruli; arterio-sclerosis is not often seen. These conditions may in some cases be late results of a specific infection that was not recognised during life.

Diseases of the Generative System, associated with ovarian activity, cause heavy losses among breeding birds, of which the commonest is perhaps simple

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egg-binding. Salpingitis and general peritonitis, due to the retention of malformed and necrosed ova in the genital tract, or to the rupture and escape of egg contents into the peritoneal cavity, are often found.

Diseases of the Liver and Gall Bladder.—Biliary cirrhosis, secondary to gall-bladder infections in birds that possess a gall-bladder, is not infrequent, and may be associated with general dropsy of the tissues, ascites and pericardial effusion. In the case of a hornbill (*Phinoplax vigil*), a gall-stone the size and shape of a date stone was impacted in the bile duct, and was associated with suppurative cholangitis and biliary cirrhosis. Fatty degeneration of the liver, often associated with angiomatous proliferation of the blood channels, is perhaps the commonest hepatic cause of death, which usually follows rupture of the enlarged and friable liver. Abscesses of the liver are generally caused by foreign bodies perforating the stomach or intestines, or they may be secondary to ulcerative enteritis. Localised necrosis of the liver is seen in parasitic infections, heterakis and coccidiosis, and other specific infections.

Diseases and Disorders of Nutrition.—Severe degrees of osteomalacia and osteoporesis are very common among young birds taken from the nest and reared by hand. True rickets with the deformity and distortion of long bones found in mammals has not yet been seen in birds at the Zoo. Gout is a common disease among elderly parrots that have been systematically overfed. It is easily recognised by the " chalk stones " deposited in the periarticular tissues about the joints of the feet and wings, and gouty spondylitis.

Neoplastic Disease.—Innocent tumours, adenomas, lipomas and granulomas are common enough, and sometimes cause death by their pressure effects. Hæmangeoma of liver and kidneys occurs, and may cause death by rupture and internal hæmorrhage. Spontaneous cancer in birds is rare. The following cases have occurred in the Zoological Gardens: A Bank Myna (*Acridotheres ginginianus*) died from an acinous carcinoma of the pancreas, with extensive secondary deposits in the liver. A Gold Pheasant (*Chrysolophus pictus*) died from an adenocarcinoma growing from the lower pole of the right kidney. The tumour occupied most of the abdominal cavity, and apparently killed by pressure effect.

Diseases of the Nervous System are seldom recognised. Cerebral abscess occurs rarely.

There are many specific infectious avian diseases, such as psittacosis, fowl cholera, pullorum disease or fowl typhoid, fowl pox, fowl plague, and others; most of them are filth diseases engendered by over-crowding, dirty aviaries, stale food and water polluted by excreta, etc. I have not seen any of these diseases in the Zoological Gardens in London, and will not describe them, as they occur mostly in domestic poultry, except psittacosis, which appears to be a disease of cage birds only.

I apologise to my readers who may have read through this meagre account of some of the more common and easily recognisable diseases of cage birds in

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the hope that they would find some useful guide or hints for treatment. Unfortunately, I am not a veterinarian, but a general pathologist, and I have had no personal experience of avian therapeutics, and have little faith in the few remedies I have seen tried. If what I have written may be some help in the recognition and prevention of certain common diseases of cage birds, my purpose will have been accomplished.

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PACHECO'S PARROT DISEASE.

By G. M. FINDLAY.

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WHILE attempting to obtain evidence of the presence of psittacosis in Brazil, Pacheco, Bier and Meyer (1930) discovered a morbid process in parrots which is due to a filtrable virus, and induces nuclear changes in affected host cells.

Rivers and Schwentker (1932) confirmed these results, and showed that the virus passes through Berkefeld V and N candles, and is restricted in infectivity to members of the parrot family, though one to two-day old chickens are partially susceptible, while chick embryos are suitable hosts for the serial passage of the virus. Man is not susceptible, and the virus is not related to that of psittacosis, though the clinical symptoms in parrots are very similar. In the liver there is fatty degeneration with areas of necrosis, while many of the affected cells, particularly in the liver, contain acidophilic intranuclear inclusions.

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RICKETS IN PARRAKEETS.

By THE MARQUIS OF TAVISTOCK.

THE phenomenon of rickets in young birds of the parrot family presents, to a person with as little medical and veterinary knowledge as myself, certain curious and unsuspected features, the season and the nature and situation of the nest having apparently far more influence than the food supplied by the parents. It should be borne in mind that the natural diet of parrakeets is of an almost

wholly vegetable nature, there being little evidence in support of the theory that in a wild state they consume numbers of grubs, etc. The young are always reared in a dark hole where no sunlight can ever penetrate.

I will now proceed with a series of data, leaving my readers to form their own conclusions: (1) Crimson-winged parrakeets (*Ptistes erythropterus*). This Australian species usually breeds in a hollow tree trunk, the eggs being laid thirty or forty feet below the entrance. A pair had one young one in a hollow tree trunk in the open flight of the aviary. It left the nest well feathered, and of fair size, but very rachitic, and quite unable to walk or fly, dragging itself slowly about by the combined use of its wings and legs. On leaving the nest it began to improve, and after some months became almost normal, and could fly strongly. It lived for many years. This bird was bred in a small fixed aviary, the parents being fed on canary, millet, oats, hemp, sunflower, peanuts, apple, and green food. Another pair, living in a large movable aviary, had a nest box with a wooden bottom inside the aviary shelter, which is a closed-in structure of matchboarding. For some reason this type and location of nest is usually fatal to all young parrakeets, and the very few that survive are generally rachitic. The majority die within a few days of hatching, but from no neglect on the part of the parents. The crimson-wings in this case reared four young, all excessively rachitic, small, hump-backed, and with twisted and deformed feet. One died soon after leaving the nest. The others were kept in a cage, and were often put in the open air. In time they improved out of all recognition, though one was never able to fly. The best was eventually able to win a prize at a rather large bird show; the second best became a strong flier, but its feet were never perfect. No special diet or treatment was given as far as I am aware.

For several years the same pair of birds in the same aviary, and with the same feeding, reared young in a natural tree trunk partly filled with damp peat and decayed wood, and standing in the open flight. One year the young left the nest perfect, and able to fly from the first. All other years they were unable to fly for a week or two on first leaving the nest, but soon became vigorous and perfect, resisting cold, keeping free from disease, and flying at liberty with great speed, and frequent displays of playfulness and high spirits. Another cock crimson-wing, paired to a Sula Island king parrakeet, produced several hybrids. Some died in the nest at an early age, but the survivors were all vigorous and perfect. They were reared in much the same type of nest as the crimson-wings. Last year this crimson-wing was paired to a fine hen of his own species. They had a bottomless wooden nest box in the open flight, partly filled with peat and decaying wood, and were given cod-liver oil soaked seed when rearing young. One nestling died when half grown; the other three were badly rickety, and did not live long after leaving the nest. This year the cock was paired to the hen of the first pair who had her old nest. Their young are perfect, and could fly from the first, but no cod-liver oil seed was given this season to any of the birds It would appear that with crimson-wings and some allied species, a natural tree

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trunk is for some mysterious reason less conducive to rickets than a wooden box in the same location, even when the material on which the nestlings rest is the same in both cases.

King parrakeet (*Alisterus cyanopygius*), an ally of the crimson-wing. Natural breeding habits similar. A pair had four young in a box with a wooden floor standing in the open flight. All were very rickety, and three died on leaving the nest. The fourth was hand-reared, being given a good deal of raw egg and milk in its food (chewed-up brown bread). It improved greatly, but was never able to fly. Another pair have reared three broods of perfect young in a nest of the type that succeeds with crimson-wings.

Amboina King parrakeet (*Alisterus amboinensis*). A single young one in the same type of nest as the common kings succeed in was rickety this year, and died soon after leaving the nest. It was deformed, and lacked the terminal joint of one wing. The male parent reached me in very bad condition, from close confinement and mismanagement, and took years to get well, so his constitution may still be impaired.

Barraband's parrakeet (*Polytelis swainsonii*), a not very distant relative of the species already mentioned. Breeds naturally in holes in trees. I have never seen a definite case of rickets in young Barrabands. They always die in nests in the aviary shelter ; always live in bottomless wooden boxes in the open flight, and sometimes live, but more often die, in nests with wooden bottoms in the open flight.

Rock Peplar parrakeet (*Polytelis melanura*), a near relative of the Barraband. Young always die if hatched in a box in the aviary shelter. I have once successfully reared perfect young in a box with a wooden bottom in the open flight, and once when the hen laid on the ground in the open. I have once lost young in the type of nest that succeeds with crimson-wings. Last year a young bird was reared in a bottomless wooden box in the open flight, its fellow dying at an early age. It had rather a pinched look, and for months could not spread and use its wings. Eventually, living in the open aviary, it became able to fly quite well, but was never a perfect specimen.

Rosella parrakeet (*Platycercus eximius*). Breeds naturally in holes in trees. An imported cock paired to an aviary-bred hen which was a poor flier, and may have been rickety in early youth, produced four extremely rickety young from a nest in the aviary shelter in 1929 ; one perfect young one from a bottomless wooden box nest in the flight in 1930 ; and (in spite of cod-liver oil seed) four very rickety young from the same nest in 1931. The same cock paired this year to a good and possibly imported hen, with the same type of nest and no cod-liver oil, had five very fine young, and one rickety one that died in the first brood, and one rickety (?) one that died in the nest in the second.

Brown's parrakeet (*Platycercus venustus*) : In a wooden box with a wooden floor in the aviary shelter, a pair had four rickety young. Two died in the nest. The other two, placed in a cage and kept in a room without vita glass, improved

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considerably without special feeding, but did not live long. In a natural tree trunk in the aviary shelter the next year, the same pair reared four good young. Last year the same cock, paired to an aviary-bred hen, had a bottomless wooden-box nest in the open flight. One young bird died on hatching, and the other at a fortnight old in spite of cod-liver oil seed. This year, in a similar nest, they reared four perfect young without cod-liver oil.

Pennant's parrakeet (*Platycercus elegans*). Some years ago a pair reared six perfect young in a nest with a wooden floor in the aviary shelter. A season or two later they lost their young in a similar nest. Last year, in spite of cod-liver oil seed, they had four very rickety young from a bottomless wooden box in the aviary flight. These soon died. This year they had two perfect young from a nest with a wooden bottom in the aviary flight. The young from this pair are themselves good breeders of strong young.

Yellow-rumped parrakeet (*Platycercus flaveolus*). In a nest in the aviary shelter a pair sometimes reared strong young, and sometimes lost them. In 1930, in their first nest, they had several rickety young ; in their second, none. The box, a bottomless wooden one, was in a very sunny part of the flight, but when the second lot of young hatched the weather was cooler and more cloudy. In 1931, in a nest of the same type, in a less sunny part of the flight, they had, in spite of cod-liver oil seed, two good young and three rickety ones. This year, in the same nest, they had nine perfect young in two broods without cod-liver oil. The young of this pair also rear strong offspring, even when mated together.

Indian ring-necked parrakeet (*Psittacula torquata*), an Asiatic species breeding in holes in trees and buildings. Three pairs of lutino-bred birds, *i.e.*, green specimens bred from a wild green cock and a wild yellow albino hen. Two cocks were perfect specimens, bred in large movable aviaries. The remainder were bred in small fixed aviaries, and show some deformity and evidence of early rickets. Pair No. I had two very rickety young in a box with a wooden bottom in the aviary shelter. These died on leaving the nest. In bottomless wooden boxes in the outside flight they have in different years reared one perfect green young one, one rather defective green young one that could not fly on first leaving the nest, but later became able to do so, and two lutino and one green young one that died soon after leaving the nest from accident or disease. Pair No. II lost one green young one in the nest last year, and reared an apparently healthy lutino, but a few days after flying it became paralysed in both legs. It improved for a time, but ended with complete use of one leg and complete loss of use of the other. It could fly strongly. Pair No. III, both defective and both bred in a fixed aviary, reared one perfect green young one in 1930, and two perfect lutinos this year. The nest was a bottomless wooden box in the flight, as was that of Pair No. II, which this year reared two moderate but not quite perfect greens.

Plum-headed parrakeet (*Psittacula cyanocephala*), a lutinistic (yellow-albinistic) wild-bred pair. Last year they had four young in a bottomless box

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in the flight. Three were very rickety, and one was normal. The parents had cod-liver oil seed. One rickety young one died; the others, kept in a cage and put out in the sun occasionally but with no special feeding, gained the use of their limbs, but died of chills during the winter. This year the parents, in a natural tree trunk in the flight, without cod-liver oil, reared four good young.

Malabar parrakeet (*Psittacula peristerodes*). A pair in a box with a wooden bottom in the aviary shelter had three rickety young. Only one survived, and made some improvement in a cage, but was never a good bird. Next year they had three good young and a mildly rickety one (which recovered) in a natural tree trunk in the aviary shelter. In 1931, they lost their young in the same nest, but in a new district where pneumonia is more prevalent.

Turquoisine parrakeet (*Neophema pulchella*), a small and delicate Australian species. Food—canary, millet, oats (dry and sprouting), grass, groundsel, and green grass seed. Nest—a little bottomless canvas box resting on a piece of thick turf in a large flight cage in a room. In 1928: one young one which developed slowly and was extremely rickety; its toes were clenched, and it was unable to close its beak. When it left the nest I supplemented the parents' feeding with raw egg and milk. In time it made a complete recovery, its beak closed, and it could perch and fly normally. In 1929 the old birds' cage was placed in a large sunny window with vita glass. They had two young, neither of which could fly on leaving the nest, and one of which was hump-backed; they were also undersized. Both improved without any additional feeding or treatment, the better one eventually growing into quite an average specimen, and laying good-sized eggs. Keston Foreign Bird Farm have hatched many turquoisines in nests with wooden bottoms in aviary shelters, and in wooden boxes without bottoms, standing outside the shelters, but with the entrance leading into them. The mortality among the young in the nest is very high, but the few that survive are always perfect.

Budgerigars are found to benefit greatly when given cod-liver oil seed in the breeding season, but the oil is not an infallible preventive of "French moult" (a disease akin to rickets in which the flight feathers do not develop properly), nor will it always stop parents with the vice of eating the feathers of their young.

The two most remarkable things about rachitic trouble appear, as I have said, to be the variation of its incidence from year to year, and the fact that with certain birds young reared in a natural tree trunk will be nearly, or quite, immune, but young reared in a board box on the same site, and on the same material (peat and decayed wood), will be badly affected. In 1931 I had about seventeen cases of rickets; this year only two or three, with much the same birds, and practically the same management. The year 1931 was a wet one, but the winter of 1931–2 was unusually dry and sunny; not that parrakeets are particularly fond of sun, for in warm weather they usually avoid it. In no case can the death or feebleness of the young birds mentioned above be attributed to parental neglect, nor to provision of insufficient green food.

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A prize cross Fox vixen belonging to H. W. MacKendrick, Wedderhill House, Blairs, Aberdeen. (By the courtesy of *Fur Farming*.)

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Nutria in their pen at the Manor Fur Farm of Mr. P. E. T. Carill-Worsley, Norwich. (By the courtesy of *Fur Farming*.)

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CAT DISTEMPER.

By G. M. FINDLAY.

Wellcome Bureau of Scientific Research, London.

THE diseases of the domestic cat have up to the present received comparatively little attention from pathologists and bacteriologists. One of the main reasons for this neglect has been the fact that it is difficult to keep cats in captivity under laboratory conditions, since they are liable to succumb to sudden and rapidly fatal epidemics of disease. Of these epidemic diseases, probably the most common is that known as " cat distemper," or, from its frequency after cats have been placed on exhibition, " show fever." Cat distemper appears to be widespread in its distribution, and while capable of infecting all varieties of domestic cat, is specially fatal to animals of the Siamese and Persian breeds. Outbreaks in zoological gardens also are not infrequent, the " lesser cats " being highly susceptible to the disease. Like canine distemper, the clinical symptoms of feline distemper are often protean. In many epidemics the symptoms are referable to the respiratory system, with coughing, sneezing, running at the eyes and nose, and conjunctivitis, either serous or purulent ; in others, there is diarrhœa and vomiting. Fever is variable. It may range from 103° to 105° F., though many animals die with typical pathological changes, but no significant rise in temperature. In some fatal cases there are no definite clinical symptoms except loss of appetite and general weakness. The mortality, especially in young cats, is high.

Ætiology.

With clinical symptoms so varied in character, it is not surprising that much doubt has existed as to the ætiology of the disease. Many varieties of bacteria have been isolated from acute epidemic diseases of cats associated with symptoms of respiratory or intestinal involvement ; Gaertner (1909), Boucek (1910), Ruppert (1912), Pospischil (1920), and Frohböse (1926), for instance, isolated organisms of the hæmorrhagic septicæmic group. In the epidemic described by Gaertner (1909) the animals refused food, began to sneeze and cough, and suffered from nasal discharge, conjunctivitis and difficulty in breathing. Pospischil (1920), on the other hand, isolated a Pasteurella from an epidemic associated with cramps, vomiting and diarrhœa, while in the epidemic reported by Frohböse (1926), the symptoms were those both of tracheitis and enteritis, the pathological changes not being characteristic of a true hæmorrhagic septicæmia. As a result there was doubt as to the ætiological significance of the bacteria isolated. Although, as shown by Hopkins and Parker (1913), cats are relatively insusceptible to streptococci, these organisms have been isolated from certain feline epidemics, notably by de Jong (1913) and Bayne-Jones (1922). In the epidemic reported by de Jong (1913), the chief symptoms were suppurative conjunctivitis and rhinitis, loss of appetite, wasting, and weakness. Fibrinous pleurisy and peritonitis were present in a considerable number of cases, while pneumonia was occasionally seen. In cats which had died from the disease, streptococci were not

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present in pure culture. In the epidemic reported by Bayne-Jones (1922) there was no pneumonia, but a greenish discharge from the nose, together with conjunctivitis, sneezing, loss of weight, and rapid exhaustion; death occurred in from four to five days.

Other organisms which have been isolated from feline epidemics with symptoms similar to those of cat distemper include members of the Salmonella group, as by Mori (1905), *Bacillus bronchisepticus* by MacGowan (1911), and various coliform organisms. Special significance has been attached by certain workers to these latter organisms in epidemics associated with acute gastro-enteritis. In view of the fact that cats, especially when young, are peculiarly liable to the invasion of secondary organisms from the intestinal tract, more convincing evidence is required of the ætiological significance of coliform organisms in feline enteritis associated with distemper. As in the case of canine distemper, which is known to be due to a filtrable virus, evidence is now forthcoming that cat distemper is also associated with an ultra-microscopic virus. Hindle and Findlay (1932) found that a filtrable virus is present in the nasal secretions and in the blood, spleen, and lymph glands of infected cats. After filtration of the nasal washings through Berkefeld N filters, it is possible to infect cats with distemper by spraying them with the filtrate, or by inoculating them, subcutaneously, intra-peritoneally or intra-cerebrally, with the blood or filtrates of the spleen or abdominal lymph glands. Death usually results about the tenth or twelfth day after inoculation, though in some cases it occurs as early as the fifth, in others as late as the thirty-sixth day. This variability would seem to depend upon the nature and virulence of the secondary bacterial invaders which almost always complicate the disease. Of three hundred cases of cat distemper investigated, only 8 per cent. were bacteriologically sterile. In all the others one or more of the following types of bacteria were present: (1) *Bacillus coli;* (2) a member of the Salmonella group, with affinities to *B. typhi murium;* (3) hæmolytic streptococci; (4) *B. bronchisepticus;* (5) para-influenza bacilli; (6) Pasteurella; (7) Streptothrix; (8) staphylococci. Cat distemper thus forms one of the group of virus diseases, including the common cold, infectious myxomatosis of rabbits, dog distemper, swine fever, swine influenza, and possibly human influenza, the clinical symptoms of which depend on a symbiosis between virus and bacteria. In swine fever and swine influenza the bacterium as well as the virus appears to be specific, while in the other diseases the virus alone is specific. In the case of cat distemper this is well shown by two epidemics which occurred at nearly a year's interval in the same cattery. In the first outbreak the only secondary organism isolated was a Pasteurella, in the second a hæmolytic streptococcus.

Pathological Changes.

The pathological changes are similar in cats dying of the natural disease, and in those infected experimentally. The most typical change consists of catarrhal congestion of some part of the gastro-intestinal tract. This congestion may range from a few small patches in the ileum to involvement of the whole length

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of the small intestine, sometimes extending into the large intestine, and even into the stomach and œsophagus. Altered blood is often present in the stomach and intestine, while congestion of the mesenteric and posterior abdominal lymph glands is common. Sometimes the cervical lymph glands also show enlargement and congestion. Congestion of the lungs, enlargement of the spleen, pleurisy with effusion, peritonitis, and occasionally hæmorrhage in the thymus are all encountered. Histologically, the changes in the intestine vary from simple congestion of the blood vessels in the submucosa to hæmorrhage, sloughing, and destruction of the mucosa. Small hæmorrhages may also occur in the muscular layer. In extreme cases the mucosa is covered by a fibrinous deposit, including red cells, desquamated epithelium, and bacteria. Infiltration with polymorphonuclear leucocytes is not a prominent feature of the intestinal lesions. The lymphatic glands often show hæmorrhages, principally in the cortex, which is the site of considerable congestion. The changes in the lungs range from congestion of the inter-alveolar capillaries, which are sometimes ruptured, to a fibrinous exudate in the alveoli. Mononuclear leucocytes are present in small numbers. The trachea is occasionally very congested, while the spleen is generally the site of active congestion, associated with the active phagocytosis of red cells. The liver and kidneys exhibit cloudy swelling. So far no specific inclusion bodies have been found.

Susceptible Species.

In addition to the domestic cat, the disease has been seen in the following species of wild cats: American tiger cat, *Felis tigrina;* African tiger cat, *F. aurata;* rusty tiger cat, *F. planiceps;* marbled cat, *F. marmorata;* caracal lynx, *F. caracal;* ocelot, *F. pardalis;* and the leopard, *F. pardus.* Lions and tigers and pumas appear to be relatively insusceptible. Dogs and ferrets are entirely resistant to infection with the virus of cat distemper, while Laidlaw and Dunkin (1926) found that cats are not susceptible to the virus of dog distemper. In addition, the disease cannot be transmitted to the mongoose, rabbit, rat, mouse, or guinea-pig. Man is also insusceptible. The infection thus appears to be restricted to members of the cat family.

Properties of the Virus.

Under natural conditions the disease appears to be transmitted either directly from cat to cat by a droplet infection, or by the infection of fomites. Soiled blankets kept at room temperature may retain their infectivity for at least sixteen days. The virus retains its activity in 50 per cent. glycerine for not less than three weeks. It is attenuated or killed after about ten days, when dried *in vacuo* at room temperature. The inoculation of such dried virus is, however, followed by some degree of immunity.

Immunity.

Cats that have recovered from one attack apparently enjoy a life-long immunity to cat distemper. While a passive immunity of short duration may be produced by the injection of large doses of immune serum shortly before exposure

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to infection, the injection of large doses of immune serum has little or no action on the course of the disease once symptoms have developed. Treatment of the disease is thus merely symptomatic. The use of bacterial vaccines may in certain cases, however, be of value provided the nature of the secondary invading bacteria can be determined at an early stage of the epidemic. In view of the encouraging results which have been obtained in vaccination against canine distemper, it seems not unlikely that a similar technique might with success be used in the struggle against cat distemper.

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NOTES ON WORMS IN FOXES.

By E. LEONARD TAYLOR, B.V.Sc. (Livpl.), M.R.C.V.S.

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IT has recently been suggested by a German investigator that the study of parasitic worms may be divided into two branches : " normal helminthology " and " pathological helminthology." Normal helminthology deals with the systematic arrangement of the numerous species, their physiology and bionomics, the side of the study proper to biologists ; and pathological helminthology deals with disease production and disease prevention, with ætiology, pathology, symptomatology, prophylactics and therapeutics ; it is more particularly the study of the medical man, and only concerns the parasitic worms which in some way are associated with disease production. The two branches cannot, of course, be clearly separated, the medical helminthologist (using the term " medical " in its wider sense to include veterinary) must know something of the classification of helminths to understand their affinities to one another, and to be able to draw comparisons between the different diseases ; and he must know something of their physiology and bionomics to be able to understand the ætiology of the diseases.

The pathological aspect of helminthology is closely associated with domestication, and the crowded conditions which agriculture and the artificial rearing of animals involves.

Crowded conditions favour the spread of all kinds of infectious diseases, but none so definitely, and none so progressively, as the disease caused by parasitic

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worms. If two animals of a standard infection are placed where one was previously kept, the ground will become twice as heavily infected, and each animal will pick up twice as many eggs or larvæ, thus leading to four times as many eggs or larvæ reaching the ground as did so in the first instance. Place three animals where there was one, and soon there will be nine times the amount of infective material reaching the ground. Place ten, or one hundred, where there was one, and the increase will be one hundred, and ten thousand respectively, giving some idea of the true comparison between conditions in the wild state and conditions under domestication.

Fox farming is one of our latest sad experiences of the harmful increase of parasitic worms where many animals are kept together. It was some time before the importance of the problem was fully appreciated, but it is now known that the control of parasitic worms is not only a major disease problem among foxes, but is a problem of first importance in fox farming, and the whole construction of the farm and the method of rearing must be planned with an eye to the control of helminthic disease. The most troublesome parasitic worms are the lungworms, the hookworms and the ascarids ; and of these, the lungworms are the most difficult problem, their situation in the host rendering medication difficult or almost impossible. Two species are involved, *Capillaria aerophila* and *Crenosoma semiarmata* ; their life-histories are probably direct in that no intermediate host is required, but the route taken by the larvæ in passing from the intestine to the lung is not known. The mature worms live in the bronchioles and bronchi, where they cause a catarrhal bronchitis, and if present in large numbers may cause complete occlusion of the lumen. The most susceptible age is from five to eighteen months, and fox cubs under eleven weeks old are not obviously affected. Many different anthelmintic treatments have been tried for the removal of these parasites, including mechanical removal with a special brush, the end being guarded by a small sponge, which is inserted into the trachea, revolved and withdrawn again, with the aim of removing some of the worms in the bristles. This treatment is only of value for *C. aerophila*, which enters the trachea much more than *C. semiaramata*, and it is claimed that by repeating this operation at intervals of three weeks all these worms may be removed, providing there is no reinfection. The beneficial results of this rather heroic treatment do not appear to have been confirmed outside the United States of America. There is also another interesting treatment, the benefit of which in a like way does not appear to have been confirmed. A secondary bacterial infection follows the parasitic worms, and it has been suggested that in some way the welfare of the worms depends upon the presence of the bacteria. A bacterium made from these organisms has been tried, and it is claimed that good results followed its use.

It is agreed by all that drugs are of no value in combating these parasites, and as with most parasitic worms, the strongest agent in control is sanitation. Treatment for parasitic worms in general is of little value even in those instances where it is effective in removing the parasite, unless sanitary measures are taken

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at the same time. Perhaps the most successful of the sanitary measures are those involving the use of specially constructed floors : wire-netting floors have given good results, but floors of wood or cement, and constructed on a slope, are more in favour at the present time. The wooden floors should be raised 18 inches or 2 feet from the ground, so that they can be kept dry, and have a slope of about six inches in twenty feet. These floors should be very thoroughly cleaned every two days ; if carefully done, this will prevent young foxes from becoming infected, and adults which already carry infection will eliminate it in about twenty months.

Foxes are good subjects for treatment with tetrachlorethylene or oil of chenopodium, and the control of hookworms is greatly assisted by regular dosing of all the animals. These drugs also have some effect on the ascarids of foxes, *Toxocara canis* (formerly known as *Belascaris canis*), or *Toxascaris leonina*. The danger from ascaris may be considered to have passed when the cubs are three months old, but although in general they are less harmful than hookworms and lungworms, they require to be kept in check, and it is considered by some observers that the pre-natal parasitism which occurs with these species is by no means a negligible cause of death.

We have known for some time that infestation with parasitic worms may take place before birth, the following species of worms having been observed occasionally to infect the young animal in this way : *Dictyocaulus filaria, Dictyocaulus viviparus, Dirofilaria immitis, Necator americanus, Ancylostoma caninum, Fasciola hepatica, Schistosoma japonicum, Trichinella spirales, Echinococcus granulosis* and *Toxocara canis*. Among this group *Toxocara canis* takes a special place, as although in the other species pre-natal infection appears to be an accidental occurrence, in *T. canis* it is considered to be the usual way in which fox cubs or dog pups become infected. This was once brought home to the writer in a surprising way at the outset of a series of experiments designed to ascertain the harm which might be done by *Toxocara canis*. A pregnant bitch was carefully washed and placed in a pen where dogs had never previously been kept. The pups were born two or three weeks later, and about two weeks after that they were given different numbers of eggs of *Toxascaris leonina*. They were then weighed at weekly intervals, and as the time approached, examinations were daily carried out to ascertain whether infection occurred. It was a surprise to find that the first eggs to appear were those of *Toxocara canis*, which had not been given to the pups at all, and that on post-mortem examination that species was present in much greater numbers than was *Toxascaris leonina*, which had been given. The infection with *Toxocara canis* had taken place pre-nataly at least two weeks previously, before the bitch had been placed in the clean pen.

In considering helminthic control in general, the necessity for preventive measures applies only to young animals, the natural resistance of adults being sufficient to protect them from harm. This particular instance would, however, appear to be an exception ; precautionary measures applying as forcibly to the pregnant vixen as to the cubs.

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The control of the parasitic worms of foxes is best maintained by constant and intelligent application of the principles of sanitation along such special lines as have been mentioned here ; it will not lead to the eradication of the parasitic worms from the fox farms, but it will keep them down to normal numbers and prevent them from increasing to pathological numbers.

SOME HEREDITABLE DEFECTS IN RABBITS.

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THE rabbit being a rapid breeder, requiring comparatively little space for housing, has been utilised as experimental material in many laboratories where funds did not permit of the keeping of larger animals. It is in many respects an ideal animal for this purpose, and has many points to recommend it. It is, therefore, somewhat remarkable that more notes have not been published of hereditable defects which have been noted. This is possibly explained in some degree by the fact that such cases are generally inherited in a recessive manner, and a stock might carry many of these without their being exhibited unless close inbreeding was resorted to. Such a practice is not usual in a mixed laboratory stock, and in the case of practical breeders for the industrial or fancy side of rabbit keeping, whilst line breeding is common, close in-breeding is generally, but not by any means always, avoided. To our knowledge close in-breeding for any length of time has only been adopted in one stock, where it was noted that many new characters made their appearance. Of these, perhaps the most striking was furlessness. In this type the young at birth are entirely bald, except for the whisker hairs, the hair later developing normally on the face and legs. The undercoat is entirely absent on the body, although the guard hairs persist. This mutation behaves as a simple recessive to normal coat, and has occurred in two stocks, one in Russia and the other at Cambridge. In the latter case the specimens have been reared and bred from, but in Kislovsky's strain they did not survive for more than one month.

A second mutation which has resulted in an effect on the fur is that known as Rex. In this the entire coat is fined down, the guard hairs losing their brittleness, and being little if any longer than the undercoat. These hairs, together with the whiskers are generally curled, and by this means Rex youngsters can be identified at birth. There is fairly good evidence for the belief that specimens exhibiting this type of fur were known towards the end of the last century, but were destroyed as undesirable freaks. About ten years ago, however, the type again appeared in France, and was retained and bred, later being exported to most parts of the world, until now it is one of the most popular varieties for fur production. The Rex coat is certainly a defect in the normal coat, and is inherited as a simple recessive to normal. It is interesting to remark that there are two, or possibly

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even three, mutations of independent genes which have produced a similar Rex coat. These are all recessive to normal coat, but when crossed together produce normals, thus proving that the characters are based on different genes.

The first Chinchilla rabbits, and more recently the first Rex rabbits, produced and imported into this country were very liable to complaints of the eye (such as in-growing eyelashes, the lids of the eye deformed, etc., and rickets. A tendency to these defects and diseases is certainly inherited, probably as a recessive character which by selective breeding may be, and actually has been, eliminated from the strain. As an example of this, the early litters of Rex rabbits could not be successfully reared without such nutritional aids as calcium, vitamins, etc., whilst now several of the leading breeders of this variety feed their stock on the same diet as other rabbits thrive on.

One interesting character which occasionally exhibits itself in Rexes is that of a complete and simultaneous moult, in which the rabbit loses all its coat at the same time, and appears completely bald. Usually the youngster succumbs to shock and cold when this occurs, but with care the animal can be reared, and future moults are normal. This type of moult appears most frequently in Rex and Rex crosses, and appears to be inherited, although the exact manner of its inheritance has not been examined yet.

The Rex coat is a "defect" which has been turned to good use by breeders on the industrial side of rabbit keeping, whilst the defect of abnormally long ears has been extenuated and treasured by breeders of exhibition rabbits. In this case the ears will measure as much as thirty inches from tip to tip, and droop downwards on the ground. This ear length is produced by the action of a series of multiple factors, and is not controlled by any single gene.

An inherited defect of some considerable importance to the fur breeder and carcase producer is that of yellow fat. The yellow colour in the fat only shows itself when stock is fed on certain types of green food, but when it does do so the carcase is not readily marketable. This character is a simple recessive to normal fat colour, and is linked with albinism. In passing, it is interesting to note that it frequently occurs in white Angoras, with the consequent result that certain of the wholesale buyers of rabbit carcases refuse to accept Angora rabbits for this purpose.

A type of torticollis, in which the rabbit permanently holds its head on one side, appears to be inherited, probably as a recessive character; whilst abnormalities such as a duplication of parts, hermaphroditism, youngsters with their noses elongated into almost an elephant's trunk, are also more prevalent in certain strains than in others, which suggests that these also are hereditable defects. It is impossible to deal fully with this interesting question in a brief paper such as this; but may we urge our readers, in the interests of the industry and of research, to report any such abnormalities as they may come across? We at Edinburgh, as well as research stations elsewhere, always welcome reports or specimens of any occurrences which may in any way depart from the normal

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THE PNEUMOCOCCAL DISEASE OF THE GUINEA-PIG.

By G. F. PETRIE.

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THE pneumococcal disease of the guinea-pig is an interesting example of an infection that attacks both man and animals; it deserves the attention, therefore, of those who are engaged upon the problems of comparative medicine. The subject, too, has a directly practical concern for every laboratory worker who has occasion to use the guinea-pig for experimental purposes, because, as we shall mention later, a quiescent infection may be set alight by various experimental procedures, and thus the results of a test may be rendered doubtful or even valueless. The disease has been known for many years, and the earliest reference to it is perhaps the paper by Binaghi (1897), an Italian worker, who described a capsulated streptococcus which he found to be abundantly present in multiple abscesses in the lungs of a young guinea-pig. Stefanski (1901) gave the first adequate description of the disease as he observed it in an epidemic among his stock of guinea-pigs in Odessa. During the past thirty years some twenty-five papers have appeared in French, German and American journals; the disease seems to have escaped the serious attention of workers in this country, if we may judge from the lack of published information. In the United States, Theobald Smith (1913–14) and Branch (1927), and in Germany, Neufeld and his colleagues, are among those who have made important studies upon it. The recent article (1932) by Professor Neufeld, of the Robert Koch Institute, Berlin, and his co-worker, Dr. Etinger-Tulczynska, should be consulted by everyone who is interested in the subject. The well-known researches on the pneumococcal group which Neufeld has carried out during many years, invest his observations on this infection with a special authority. The present writer has been familiar with the disease since October, 1924, when an outbreak occurred among the breeding stock of guinea-pigs in the Serum Department of the Lister Institute at Elstree. The account which follows is drawn from his own experience, and from the published contributions to the subject.

The Age and Sex Incidence.

The disease is chiefly incident upon females, and this is certainly true when a breeding stock is under observation, because pregnancy is a powerful predisposing influence. For this reason also adults are specially affected. The younger reserve stock before being mated appear to be definitely less susceptible to the infection—a fortunate circumstance for those who are engaged in the production of therapeutic sera and who use guinea-pigs for the titration of diphthéria and tetanus antitoxin, because animals of the requisite weight for testing—250 to 350 grams—are seldom attacked. The young may die of the disease within the first three weeks of life, but if they survive beyond the fourth week they are

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unlikely to acquire a fatal infection before reaching adult life (Petrie, 1930). Adult males in the breeding stock are liable to contract the pulmonary form of the disease.

The Symptoms.

The symptoms are those which accompany any acute or chronic infection. The animal remains quiet, with humped attitude and ruffled fur, but the most characteristic symptom is dyspnœa. A wet snout from nasal catarrh is an important sign of the infection. The disease may assume two forms: (1) a respiratory form with nasal catarrh, or even a pneumonia; and (2) a uterine infection. Both may co-exist, and the symptoms are referable to all degrees of an acute or chronic infection. The disease in its most acute form is septicæmic, and thus widely scattered lesions may be found on post-mortem examination. In Neufeld's experience a symptomless carrier state is also commonly found. An interesting clinical sign which the present author has not infrequently noted is the occurrence of petechiæ in the ears of newly-born or quite young animals. The stock at Elstree consists in a high proportion of white or cream-coloured guinea-pigs, so that the hæmorrhages are readily observable. They are caused by a purpurogenic toxin, which is common to all types of the pneumococcus, and which hitherto has been known only to be capable of producing hæmorrhages, as an experimental lesion, in white mice. (Julianelle and Reimann (1926, 1927), and Mair (1928)).

The Post-Mortem Appearances.

A good account of the lesions will be found in a paper by Branch (1927). Here it will suffice to note that the appearances differ according to the type of the disease. In the pulmonary form the lungs may be extensively affected, with hepatised portions that sink when they are placed in water. A sero-fibrinous and hæmorrhagic pleurisy with an abundant effusion is often seen, and an exudative pericarditis is, too, not uncommon. The uterine lesions are confined to the breeding sows, and are associated with pregnancy, so that a common occurrence is for an animal to give birth prematurely to dead fœtuses, or to produce living young, and to succumb within a few days, when obvious signs of a uterine infection will be apparent post-mortem. In such an event a pneumococcal peritonitis is often found. A pale, fatty-looking appearance of the liver is a characteristic sign, whatever may be the type of the infection.

The Causative Agent.

Strains of the guinea-pig pneumococcus which were isolated at Elstree some years ago were pronounced by Dr. Mair and by Dr. F. Griffith to fall into the heterogeneous Group IV. Neufeld noted that all his strains were serologically identical. He sent some of his agglutinating serum and one of his strains to Dr. F. Griffith, of the Ministry of Health laboratory in London, who carried out tests with samples of the type sera which he had received from Miss Cooper of New York. In recent years Cooper and her co-

workers have isolated some thirty serological types from cases of pneumonia in man, in addition to the three original well-known types, and have prepared agglutinating sera from them. Dr. Griffith was able to identify the Berlin strain as Miss Cooper's Type XIX. In October and November, 1931, we sent some of the Elstree strains to Dr. Griffith, and he found that they, too, were Type XIX. We then prepared an agglutinating serum from an Elstree Type XIX strain, and were thus able to determine that every strain we isolated—some two dozen—conformed to this type.

In a recent paper, Cooper, Rosenstein, Walter, and Peizer (1932) state that Type XIX was isolated from six cases of lobar pneumonia of adults, of which five were severe and one was of moderate severity ; a patient with a bacteriæmia died. This type was also isolated from seven cases of lobar pneumonia in children, four of which were moderate or mild. Type XIX was found to be fairly prevalent in normal persons. The strains were of medium virulence for mice. Gundel and Schwarz discovered Type XIX in man on a good many occasions, and Neufeld and Etinger-Tulczynska have ascertained it to be one of the most frequent types of pneumococcus in the nasal catarrh of infants. They have also proved it to be the cause of a spontaneous pneumococcal outbreak in laboratory rabbits. It is natural to suppose that the infection may be passed on to a stock of guinea-pigs by the animal attendants, but no proof of this has as yet been adduced. Cultures from the throats of the regular attendants at Elstree proved to be negative in this respect. There are a few scattered references to other types of pneumococcus as the causal agent in spontaneous infections in guinea-pigs, but the details are scanty. Type XIX certainly appears to be the most important type in the natural disease.

General agreement exists among those who have tested their cultures on guinea-pigs, mice and rabbits, that although an experimental infection may succeed in these species, the strains are of low virulence on the average. The results that are given in the older papers may be discounted to some extent, owing to ignorance of the change, by continued subculture without animal passage, of the smooth, recently-isolated strains into the rough avirulent modification. The cocci are usually present in large numbers in the chief lesions on microscopical examination. A capsule stain should always be employed as a diagnostic reagent. Hiss's stain gives very satisfactory results. The guinea-pig strain possesses a well-developed capsule. For identification of the type Neufeld advises the method he described many years ago, namely, the addition of undiluted specific serum to the culture or the fresh material from the guinea-pig, and the observation that within a short time the homologous cocci become characteristically swollen on account of an antigen-antibody interaction in the capsule.

The Epizootiology of the Disease.

The Carrier State.—Selter, in the year 1906, published observations which showed that pneumococci could be isolated from the lungs of apparently healthy guinea-pigs. Neufeld and his co-worker, in their recent paper, furnish ample

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confirmatory evidence of the existence of carriers which harbour dormant infections in the upper respiratory tract. They found the specific pneumococcus in the nasal cavities of 55 per cent. of 329 animals ; in many of them the infection persisted for months. A smouldering infection of this type, as well as the acute pulmonary form of the disease, clearly offers abundant opportunities for contact infection by droplets of infective material, and in other ways. Whether a severe infection will supervene in the recipient or not depends, in the opinion of Neufeld and other German authors, upon its disposition or constitution, that is, the obscure factors which are concerned in natural immunity.

Predisposing Causes.—The comparatively feeble virulence of the infecting strain of pneumococcus, and the difficulty of bringing about contact infection under experimental conditions, have led to the view that a naturally implanted infection is not likely to result in an acute process unless some predisposing factor plays a part. Several factors have been discussed, such as, for example, fluctuations of temperature in the animal house, exposure to severe cold, dampness or draughts, a lack of one or other of the vitamins in the food, and pregnancy. In the view of the present author, all of these influences may lessen the individual resistance, and thus favour the spread of the infection. The outbreak in October, 1924, at Elstree, was almost certainly attributable to a deficiency of vitamin C in the diet, because at that time an adequate ration of green food was not available. When an ample supply of green food was given, the health of the stock showed a marked improvement, and the mortality from the pneumococcal infection and from all causes was considerably reduced. Nevertheless, the mortality, especially among the newly-born and very young animals, tended to increase in the first quarter of the year, a seasonal relation which suggested the exhaustion during the first half of the winter of a vitamin reserve in the breeding sows that had been stored up in summer, with a consequent lowering of the resistance of both parent and young to the attack of the pneumococcus. A deficiency in vitamin D was considered as a possible factor, and accordingly an experiment was undertaken throughout a year, whereby half the breeding stock were irradiated by means of a mercury-vapour lamp ; the remaining guinea-pigs served as a control group. However, an analysis of the health conditions in the two groups furnished no evidence of any benefit having been conferred on the irradiated animals (Petrie, 1930). The influence of pregnancy has been remarked upon by a good many authors, and particularly by Richters (1913). This has been the dominant feature of the infection at Elstree, doubtless because the guinea-pig population here is essentially a breeding stock. The circumstance which has been referred to by the German workers, and by Nicolle, that the experimental inoculation of various toxic agents or bacterial cultures, and the induction of experimental scurvy are often followed by a frank pneumococcal infection, gives support to the view that debilitating influences are apt to set alight a latent focus.

Seasonal Prevalence.—A review of the articles that have been written on the pneumococcal infection of guinea-pigs makes it clear that the disease is mainly

prevalent during the winter and the early spring months. The reason for this is apparent from the considerations that have just been set forth. A shortage of green food and unfavourable atmospheric conditions undoubtedly lower the general level of resistance in a guinea-pig population.

The Prevention of the Disease.

The arguments that have already been advanced make it unnecessary to do more than briefly comment on this aspect of the problem. Neufeld advises a search for carriers by means of a special technique, of which he gives a description, and the elimination of such animals from the stock. Without doubt this is the ideal method at which to aim, but the method may not always be easy to adopt in practice. Successful attempts at vaccination have been reported by a few workers, for example, Chevrel and his associates, and Bruckner and Galasesco in Bucharest. The guinea-pig stock at Elstree has been actively immunised on several occasions by means of vaccines prepared in various ways, but the results, as far as they can be judged, were quite disappointing. Neufeld and his colleague have not up to the present obtained convincing evidence of the utility of the method as a means of reducing the incidence of the disease, and they consider that further investigation in this direction is desirable.

The suggestion was made to us by Miss E. M. Hume that the addition of iodine to the diet might increase the resistance to the infection. Accordingly, during 1930, half the stock were given 0.04 mgm. potassium iodide per animal each day; the remaining animals were kept as controls. The results have not yet been accurately analysed, but we have no reason to believe that the infection was modified in any way by this treatment.

Acting on the supposition that an abundance of fresh air and a process of " hardening-off " in the open air, even in the winter, might increase the resistance to the infection, the present author carried out an experiment of this kind, but the results were neither beneficial nor harmful.

We have gained the impression from the experience at Elstree that the disease may be effectively controlled if the comfort of the stock and the cleanliness of the pens are regularly maintained, and if an abundant and nutritious diet, which includes plenty of green food, is provided throughout the year. During February and March of the past year a recrudescence of the infection occurred as the result of an unavoidable shortage of green food in the diet. When the ration again became normal the infection ceased, and during the past six or seven months illness and death from the disease have virtually disappeared. We believe that if it were possible during the winter to maintain artificially the summer conditions of climate and food supply, the infection would die out altogether, at least in its acute form; for it is permissible to doubt, for example, whether the winter vegetables have the same food and vitamin value as the actively growing green plants that are irradiated by sunlight in the summer.

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We possess at Elstree a complete record of the history from birth of every guinea-pig in the breeding stock. This system has been in operation for some years, and it enables us to reserve for the breeding stock the progeny of sound parents. The mother must show a clean record of litters, and her previous litters must have been of average size—not more than four or five—because we do not think it desirable to encourage the production of litters of six, seven, or eight young. The average weight at birth of guinea-pigs in large litters is small, and a large family puts a strain upon the mother during the lactation period; thus the young are not likely to thrive so well as animals from litters of average size. An analysis has already been made of the records during a period of fifteen months, when the irradiation experiment was in progress. It is hoped to extend the analysis in order to obtain exact information on the fertility data, the general health conditions of the stock, and the incidence and distribution of the pneumococcal infection.

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THE TRANSMISSION OF TUBERCULOSIS BY CONTACT FROM INFECTED TO HEALTHY GUINEA-PIGS, WITH REFERENCE TO SOME RECENT WORK ON B.C.G. VACCINE.

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In view of the vast amount of work on tuberculosis which has been carried out on guinea-pigs, it is strange that there should remain any difference of opinion as to their susceptibility to natural infection.

Calmette (1922), in his book on tuberculosis, makes the following statement: "Le cobaye ne prend jamais ou presque jamais spontanément la tuberculose. Dans les élevages, même lorsque ceux-ci sont effectués dans des locaux dépendant des laboratoires, il est tout à fait exceptionnel de trouver un des ces animaux contaminés. Par simple cohabitation dans des cages, ou dans des parcs avec des animaux artificiellement tuberculisés, il est très rare qu'ils s'infectent."

Remlinger (1923), who carried out extensive experiments on this question, was unable to confirm the statement of Calmette; he found that healthy guinea-pigs contracted infection when kept in the same cages as tuberculous animals, but he never observed instances of the infection passing from one cage to another.

Schroeder and Crawford (1929) found that healthy guinea-pigs contracted infection when kept for a considerable period in the same house, but in separate cages to infected guinea-pigs. They add that there are records of several cases of spontaneous tuberculosis occurring among guinea-pigs at their Institute.

A. S. Griffith (1930) pointed out that the liability of the guinea-pig to contract tuberculosis spontaneously was noted by Robert Koch (1884), and has since been confirmed by many workers. During the period 1912–1927, Griffith observed seven instances of spontaneous tuberculosis in guinea-pigs ; examination of the strains showed six to be of bovine, and one of human origin. He recorded also two cases of spontaneous tuberculosis in guinea-pigs caused by an avian strain of tubercle bacillus ; this would appear to be the first reported case of spontaneous avian tuberculosis in the guinea-pig. The evidence pointed to infection having resulted from a tuberculous pigeon kept in an adjoining room.

In the course of experimental work on B.C.G. vaccine, Watson, McIntosh and Konst state that they have observed healthy guinea-pigs do not contract infection when placed in the same cage as tuberculous guinea-pigs. Their statement is as follows : " Furthermore, we have had occasion to record the interesting fact that normal, healthy guinea-pigs, when placed as control animals in the same cage with tuberculous guinea-pigs, and surviving the latter, appear to escape infection, as no lesion could be found in them when killed." This observation is contrary to general experience, and would not have been of more than passing interest were it not for the fact that the authors claim that in guinea-pigs inoculated with B.C.G. vaccine, 30 per cent. showed tuberculous lesions, and 9 per cent. developed " extensive caseating generalised tuberculosis."

The following is a summary of the results of the pathogenicity tests which they carried out : " No lesions discernible in 94 guinea-pigs (average duration 192 days)." " Early, slight, or localised in 32 guinea-pigs (average duration 290 days)." " Typical tuberculosis in nine guinea-pigs (average duration 309 days)." They mention that their experimental guinea-pigs were kept in a separate house.

It is a noticeable fact that only one guinea-pig died of tuberculosis within 150 days of inoculation with B.C.G. They give as a possible explanation of these results that " virulence is a latent property of B.C.G., requiring some unusual stimulus, and especially time to awaken it to activity," and they explain the failure of other investigators to obtain similar results by the fact that most of the observations on guinea-pigs have been made within five months of inoculation with the vaccine. At the time their article was published, that statement was probably correct, but since then many laboratories have held B.C.G. vaccinated guinea-pigs for long periods without being able to confirm the Canadian results. Since the observation of Watson and his co-workers, some hundreds of guinea-pigs inoculated with B.C.G. vaccine in this laboratory have been retained for one year, and none has shown on autopsy any trace of tuberculous infection. The consensus of opinion is that B.C.G. vaccine is innocuous. It is unnecessary here

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to mention the numerous authorities upon which that statement is based, and it will suffice to mention that A. S. Griffith, in an important contribution to the subject, recently expressed the opinion, based on extensive feeding and inoculation experiments, that B.C.G. vaccine was incapable of producing progressive tuberculosis in rhesus monkeys, which, he pointed out, are the most susceptible of all mammals to infection with mammalian tubercle bacilli.

It is the custom in many laboratories to house B.C.G. inoculated guinea-pigs away from other stock in order to eliminate any possibility of accidental contamination, so if the observation of the Canadian workers is correct, this would appear to be an unnecessary precaution. In order to test the accuracy of the observation that normal guinea-pigs do not contract infection when held in contact with tuberculous guinea-pigs, the experiment here described was carried out. Fifty-six guinea-pigs were used: twenty of these were artificially infected, and the remainder kept as controls. The experiment was arranged in the following manner: Three healthy guinea-pigs were placed in each of eight cages (18×13×10 in.) with artificially infected animals. These cages were arranged in two rows of four each, and smaller cages (11×11×9 in.) each containing two healthy guinea-pigs, were interposed between the large cages (see ground plan).

Ground Plan Showing Arrangement of Cases.

SUBCUTANEOUSLY.

Cage No.	**1**	Small Cage	**2**	Small Cage	**3**	Small Cage	**4**
Infected Guinea-pigs ...	1		2		3		4
Healthy Controls ...	**3**	**2**	**3**	**2**	**3**	**2**	**3**

INTRAPERITONEALLY.

Cage No.	**5**	Small Cage	**6**	Small Cage	**7**	Small Cage	**8**
Infected Guinea-pigs ...	1		2		3		4
Healthy Controls ...	**3**	**2**	**3**	**2**	**3**	**2**	**3**

Light type denotes infected guinea-pigs
Heavy type denotes controls

In order to vary the degree of infection to which the control guinea-pigs were subjected, a different number of infected animals was placed in each cage: thus there was one infected animal in Cage I, two in Cage II, three in Cage III, and four in Cage IV. The artificially-infected animals in one row of cages were inoculated subcutaneously, and in the other row intraperitoneally, with 0.5 c.c. saline suspension of spleen obtained from a guinea-pig affected with tuberculosis. The guinea-pigs infected intraperitoneally died in an average time of 42.8 days, while those inoculated subcutaneously died in average time of 55.7 days. The experiment lasted for 233 days, and the in-contact animals which remained alive at the end of that period were killed and examined. All deaths attributed to tuberculosis among the controls were confirmed by microscopic examination, by isolation of the organism, and in the majority of instances by guinea-pig inoculation.

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One of the Silver Foxes at Stack's Fox Farm, Detling, Maidstone. (By the courtesy of *Fur Farming*.)

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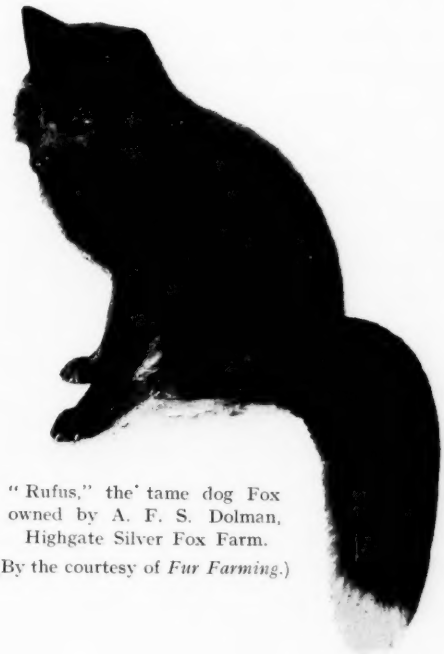

"Rufus," the tame dog Fox owned by A. F. S. Dolman, Highgate Silver Fox Farm.
(By the courtesy of *Fur Farming*.)

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Controls in Contact with Guinea-pigs Infected Subcutaneously.—Three of the twelve control guinea-pigs died of causes other than tuberculosis. Eight controls (66 per cent.) died of tuberculosis between the 96th and the 132nd days. One guinea-pig killed on the 233rd day was found to be healthy.

Controls in Contact with Guinea-pigs Infected Intraperitoneally.—Four controls (33 per cent.) died of tuberculosis between the 99th and the 176th days. The remaining eight controls were killed on the 233rd day, and found to be healthy. None of the guinea-pigs in the small cages died during the experiment, and none showed lesions of tuberculosis when autopsied on the 233rd day.

It is clear from the results obtained in this experiment, which confirm those of many other workers, that healthy guinea-pigs contract infection when held in contact with tuberculous guinea-pigs. It is of interest to note that in most of the recorded cases of spontaneous tuberculosis among guinea-pigs the disease evolved slowly, and this may partly explain the relatively few cases observed, as guinea-pigs are not usually held in experiments for long periods. In view of the observations of Robert Koch, Griffith, Bruno Lange, Kolle, and others, there can be no doubt but that indirect infection (so-called spontaneous infection) does occur among guinea-pigs, and probably more frequently than is generally recognised. As it is now established that B.C.G. vaccine does not give rise to progressive tuberculous lesions capable of sub-inoculation into susceptible animals, it is probable the tuberculous infection in guinea-pigs inoculated with B.C.G., observed by the Canadian investigators, arose spontaneously, and in support of this view is the fact that only one guinea-pig died of tuberculosis within 150 days of inoculation with the vaccine.

Conclusion.

(1) Sixty-six per cent. of normal guinea-pigs contracted tuberculosis when held in the same cage as guinea-pigs infected subcutaneously with bovine tubercle bacilli. (2) Thirty-three per cent. of normal guinea-pigs contracted tuberculosis when held in the same cage as guinea-pigs infected intraperitoneally with bovine tubercle bacilli. (3) None of the guinea-pigs in this experiment contracted tuberculosis by indirect contact, but numerous recorded observations prove that this occasionally occurs.

SUMMARY.

Cage No.	Number of artificially infected guinea-pigs in cage.	Route of Infection.	Healthy in-contact guinea-pig.			Post-mortem Examination.
			No.	Result.	Days.	
1	1	Subcutaneously	43	Died	57	Negative
		,,	44	,,	216	,,
		,,	45	Killed	233	,,
2	2	,,	46	Died	112	Tuberculosis
		,,	47	,,	120	,,
		,,	48	,,	131	,,
3	3	,,	49	,,	132	,,
		,,	50	,,	160	,,
		,,	51	,,	29	Negative

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Cage No.	Number of artificially infected guinea-pigs in cage.	Route of Infection.	Healthy in-contact guinea-pig. No.	Result.	Days.	Post-mortem Examination.
4	4	Subcutaneously	52	Died	96	Tuberculosis
		,,	53	,,	96	,,
		,,	54	,,	155	,,
5	1	Intraperitoneally	55	,,	99	,,
		,,	56	Killed	233	Negative
		,,	57	,,	233	,,
6	2	,,	58	,,	233	,,
		,,	59	,,	233	,,
		,,	60	Died	156	Tuberculosis
7	3	,,	61	,,	113	,,
		,,	62	,,	176	,,
		,,	63	,,	233	,,
8	4	,,	64	Killed	233	Negative
		,,	65	,,	233	,,
		,,	66	,,	233	,,

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SOME ECONOMICS OF RABBIT FUR AND WOOL PRODUCTION.

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THE breeding of rabbits in confined areas has been carried on in different parts of the world from remote times. In England we know from reports made to the Board of Agriculture (about 1805–18) that rabbits were cultivated in most parts of the country. Middlesex appeared to be notable, for the rabbits of that county were mainly kept in small hutches. Rabbits have been bred in many European countries from early times. Belgium and France have been specially conspicuous by their rabbit-keeping activities, and have latterly shown a pronounced bias towards table rabbit production.

Breeders in England have been keen competitors at live-stock shows for nearly fifty years. With the post-war expansion of fur and wool production, a great impetus was given to rabbit showing, and there were, and are, many hundreds of these show competitions held annually. Showing has resulted in comparatively rapid improvement of quality in the newer fur breeds, and England has supplied large numbers of pedigree breeding animals to numerous foreign and colonial countries which have recently taken up rabbit keeping. The sale of

breeding stock is an important matter with many of the leading British breeders. At the 1932 Harper Adams College Rabbit Conference, Dr. McDougall[1] admitted that when money was plentiful they actually sold over £7,000 worth of live rabbits in just over six years. To-day when money is less freely spent, there are many pedigree rabbit breeders who find the sale of livestock well worth while. It is mainly these breeders who are so keen on improving the different varieties, and in the course of achieving this result they become the main supporters of shows. During the year 1931 there were just over 65,564 entries of live rabbits at 334 of the open shows held up and down the country. This is exclusive of the vast number of small or purely members' shows, and these latter may secure as great a total entry as the open shows. At one show, *viz.*, Bradford, there were 3,351 entries of live rabbits.

Imports and Exports.

Large numbers (about 50,000,000) of raw or treated rabbit skins are imported into the United Kingdom each year; there are also appreciable re-exports and exports of home produced skins. The bulk of this trade is in skins of low value, but these indirectly affect the trade in better quality British tame skins of selected varieties. We also import upwards of a quarter of a million hundredweights of fresh, frozen, or preserved rabbit meat per annum, nearly all of which is consumed here. In addition, there are imports of Angora rabbit wool to the extent of nearly one and a-quarter thousand hundredweights. No figures are available to show the imports or exports of articles partly or wholly manufactured from rabbit fur or wool. Particulars relating to the external trade in live rabbits are also lacking. Below are set out the available details for the year 1930.

IMPORTS AND EXPORTS OF RABBIT PRODUCTS,[2] 1930.

	Imports.		Exports.	
Meat.	Cwt.	£	Cwt.	£
Fresh ...	60,937	271,263	14	52
Frozen	228,704	473,959	9,572	22,015
Tinned, etc. ...	3,377	13,163	175	801
Total meat	293,018	£758,385	9,761	£22,868
Skins.	No.	£	No.	£
Dressed	14,530,432	914,396	2,107,026	137,086
Undressed ...	37,965,778	638,852	77,522,492	1,358,674
Total skins	52,496,210	£1,553,248	79,629,518	£1,495,760
Wool.	Cwt.	£	Cwt.	£
Raw	1,240	120,271	9	746
Grand Total		£2,431,904	... ...	£1,519,374

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Produce Prices.[3]

Rabbit skins of selected varieties such as Chinchilla, Havana, Beveren, and others, have been sold through fur rabbit breeders' co-operative societies (such as the Fur Board), by public auction, and by private trading. From the returns of one large fur auction company it is seen that 162,628 raw skins of four breeds were sold during the period 1927–31. The average prices for the different varieties ranged from 2*s*. 1*d*. to 3*s*. 7*d*. each, the best average being 7*s*. 6*d*. each for Havanas in 1928. By private trading from a demonstration and educational centre we have sold dressed skins at prices which have varied in the different years (1927–32) from an average of 10*s*. 2*d*. to 4*s*. 6*d*. each for recognised fur varieties. During the years 1926–30, we sold the carcases, dressed by various methods, and obtained an average price of just over 8*d*. per lb. In 1930–32, the trade improved, and the average price for carcases dressed in the " Ostend " style rose from 9*d*. to 11*d*.–1*s*. per lb. The *Weekly Marketing Report* issued by the Ministry of Agriculture includes notes on the rabbit trade, and it is seen that wild rabbits sell at little more than half the price per lb. obtainable for good tame rabbit carcases.

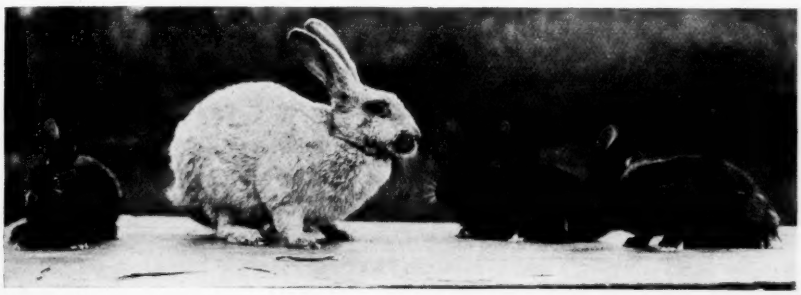
Angora rabbit wool has been sold at prices which averaged 34*s*. 3¾*d*. to 29*s*. 1¾*d*. per lb. from 1926 to 1928. Since then the trade appears to have been placed on a more commercial basis, and the average price per lb. during 1929–32 has been 25*s* 5½*d*. to 23*s*. 2½*d*. per lb., under a system where each of our adult woollers has been brushed, and then clipped, only four times per annum. Breeders who groom more frequently are able to obtain a somewhat higher average price for Angora rabbit wool.

Health and Research.

The rabbit is a notoriously prolific animal. With an average litter size of about 5–5.5 young, and two to three litters per annum, young does are frequently ready for breeding at six months of age. This amazing rate of increase raises numerous problems for the large-scale rabbit keeper. A well-known breeder[4] with a stud of about 5,000 head has written of the dangers of trying to rear large litters on mass lines. When up to nine young were left with the doe, over 20 per cent. died, and of the survivors only a very small percentage did at all well. Much better results were obtained when the following procedure was adopted: litters born up to May, allow six young per doe; litters born up to June–July, allow five young per doe; litters born up to August-September, allow four young per doe.

Breeders who have small studs, and feed their animals as individuals, are usually able to rear large litters without much difficulty; but at the present time there are numerous people who restrict the number of young in the litter to about five, and thus usually obtain good results. There is a vast field for research into problems relating to body and coat growth, feeding, reproduction, and health, in the rabbit. County instructors and National Rabbit Council advisers are restricted in their activities owing to the comparatively limited knowledge of successful prevention and treatment of diseases of the rabbit. At the present

FIG. 3. A smear preparation of a mycetoma (*Penicillium*) growing within the syrinx of a Bird of Paradise (× *ca.* 800).
(Article by A. E. Hamerton, M.R.C.S., *page* 5)



Argent de Champagne Fur Rabbits at the Harper Adams Agricultural College. This variety is peculiar in that the first baby coat is black, and when this is shed the succeeding coat is the colour of old silver.
(Article by W. King Wilson, *page* 34)

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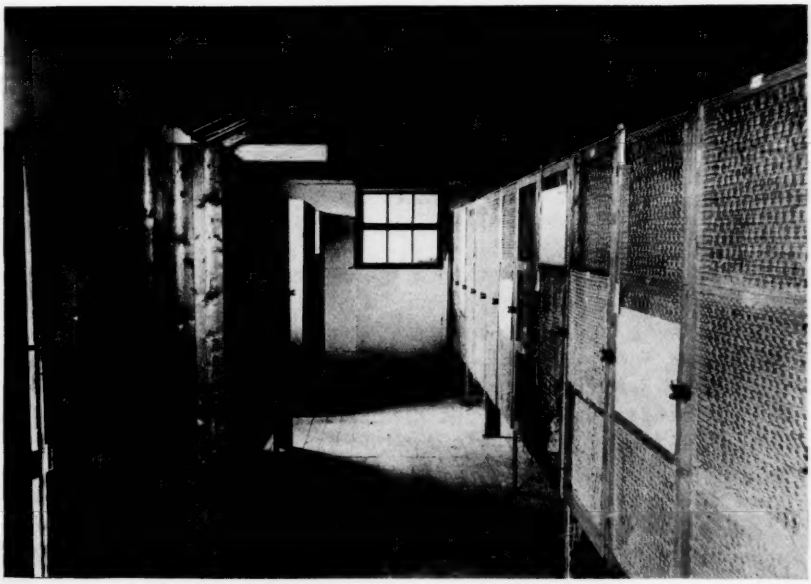

The Rabbit Department at Harper Adams College.
(Article by W. King Wilson, *page* 34)

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time few veterinary surgeons are able to prescribe suitable treatment for sick rabbits. When members of the veterinary profession can successfully prescribe for the rabbit there is little doubt that a large number of breeders will make use of their services.

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SOME POISONOUS PLANTS OF KENYA.

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THE six plants described in this paper were collected while the writer was at Kabete, Kenya, over the years 1927–1929. They were either collected in the laboratory paddocks, or else obtained on farms where suspected cases of plant poisoning were under investigation. Some of them, *e.g.*, Nos. 3, 4, 5 and 6, are new to toxicological literature, though, as mentioned in the text, two species of No. 4 have already been experimented with in Australia.

Of all the plants mentioned in the present paper, No. 6, *Stephanorossia palustris*, is the most important on account of its wide distribution and high toxicity at certain seasons of the year. No attempt could be made, unfortunately, to find out the poisonous agent of the plant, though on every occasion steps were taken to exclude hydrocyanic acid by the simple picrate paper test.

I have to thank Mr. A. D. Cotton, of the Royal Botanic Gardens, Kew, Richmond, Surrey, for the identifications of the plants described in this paper.

Plant No. 1.—Cucurbitaceæ. Cucumis ficifolius.

Vernacular names: Wild gourd (colloquial); Kikungwe (Kikuyu); maneraria (Lumbwa).

Occurrence.—A very common xerophytic scandent plant readily recognisable by its yellowish, gourd-like, bluntly tuberculated fruits, which at first are green in colour, turning yellow as they ripen. Its stems and coriaceous leaves are richly studed with numerous sharp curved spines. The plant grows conspicuously in the grazing, and appears to be severely left alone by stock, but in places it may be covered by other vegetation, and may be more difficult to see. The writer has observed it in rank grass cut for litter, and it is probably either in this way, or mixed with hay, that it is mostly eaten by cattle and smaller stock. It grows well on land that has been allowed to get weedy or uncultivated. The Kikuyu and Lumbwa both reported the plant as highly poisonous.

Literature.—In South Africa both *Cucumis africanus Linn* and *C. myriocarpus Naud* have been shown to be very poisonous, though the active principle

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of both plants has not yet been determined. Quin[1] is of the opinion that it is "probably of a resinous nature." In Western Australia, *C. myriocarpus Naud* is an introduced noxious weed, and is regarded in that part of the continent as being harmful. Here it is known colloquially as "paddy melon," "wild melon," "prickly cucumber," etc. Cattle and sheep[2] are said to eat the melons in large amounts without ill-effect. In New South Wales[3] the continued ingestion of the melons of this plant has been blamed for causing blindness in horses. Experimental evidence shows that the blindness cannot be associated with the feeding of this plant, though ingestion of the vine itself leads to impaction of the colon. No reference in the literature can be found to the species described in this paper.

Symptoms of Poisoning.—As the animal refused to eat the plant, drenching was relied upon. Finely chopped up leaves, stems, flowers, and fruits were used. The first drench of 300 grams was not followed by any symptoms, but a second drench of the same amount on the following day resulted in great depression, salivation, and foaming at the mouth, grunting, congestion of the visible mucosæ, and profuse liquid green diarrhœa. As the poisoning progressed, there was considerable moaning, and increased respiratory distress. The diarrhœa increased hourly in amount, becoming watery, very fœtid, and olive-green colour. The animal weakened rapidly, became more or less completely prostrated, and before death passed into a state of coma. These peracute symptoms were produced by a total of 600 grams of the plant. In the second case poisoning followed the administration of 900 grams of material over three days. The symptoms were the same in both cases, and were obviously referable to an acute gastro-enteritis.

Pathological Anatomy.—The most noticeable feature at autopsy was acute hæmorrhagic gastro-enteritis, complicated by severe ulceration of the gastric mucosa. Other changes recorded were acute congestion and œdema of both lungs, a dilated flabby heart, and acute congestion of liver, kidneys and spleen.

Result of Test.—*Cucumis ficifolius* is a very toxic plant for sheep. When drenched in amounts of 600 grams and 900 grams to two animals, it rapidly caused death from acute gastro-enteritis. This plant is undoubtedly one of the most deadly yet found in East Africa. No attempt was made to determine the most toxic part of the plant. Quin[1] records that the green fruits of the commoner South African species are far less poisonous and less bitter than the ripe ones. In his experiments he relied entirely on fruits, the juice from 1 kilogram of ripe fruit killing a sheep in six hours, while that from 2½ kilos of green fruit only produced a mild diarrhœa. In the experiments described in the present paper fruits in all phases of maturity were employed.

Plant No. 2.—Ranunculaceæ. Ranunculus pinnatus Poir.

Vernacular name: Wild buttercup (colloquial).

Occurrence.—A very common erect herb in swamps, rivers, or marshy places throughout tropical and sub-tropical Africa. The same plant, under the name *R. pubescens Thunb*, has been described as poisonous by several writers in the

Union of South Africa. At one time it was suspected of being responsible for the so-called vlei poisoning described by Neser and Mogg.[4, 5] It is easily recognised as belonging to the buttercup family by its characteristic deeply divided leaves, and determinate yellow inflorescence. In the Kabeter district of Kenya this plant is very common, and flowers all the year round. It is said to possess a bitter acrid taste, and to be avoided by stock. The natives regard it as highly poisonous.

Literature.—Veterinary toxicology is rich in poisonous species of ranunculus, which have been recorded from many parts of the world and from different climates. In Great Britain alone species known as spearwort, crowfoot, lesser celandine, and the buttercups have all been found to be poisonous. In South Africa, Le Roux[12] proved the toxicality of *R. pubescens* (*R. pinnatus*) at Ermelo (N.E. Transvaal), a finding that has since been confirmed by Steyn,[6] with material from Ermelo and the Pretoria districts. Both Le Roux and Steyn used sheep in their tests, but Neser and Mogg[4, 5] state that *R. pubescens* (*R. pinnatus*) has not proved toxic at all for bovines when fed at the rate of 2 lb. daily. From their feeding tests they exclude any rôle of this plant in the ætiology of vlei poisoning. There are no records of the East African plant ever having been shown to be poisonous, though several farmers regarded it with suspicion, probably as the result of their acquaintance with allied species in Britain or South Africa.

Symptoms of Poisoning.—One thousand grams were sufficient to cause symptoms of illness in sheep, though larger amounts (1,800 grams) were required to cause death. In the present test the fresh flowering plant was fed with green lucerne, and was apparently all eaten. In one sheep (No. 2,766) there was a sharp rise of temperature after 1,500 grams had been eaten. The fever was in the region of 104.8° F. for seventy-two hours, but fell to 102° F. at the time of death. The chief symptoms of poisoning were profuse ropy salivation and trembling of the lips, which were found to be associated with acute ulcerative stomatitis. The conjunctiva was congested, and the animal stood in one place with its head depressed, and moaning. There was very marked diuresis ; anorexia was complete, but no diarrhœa was noticed. The fæces became very soft and compact, completely losing their pellet-like form and shape. Even before death there was no diarrhœa, the most striking symptoms being those associated with acute ulceration of the buccal mucosa.

Pathological Anatomy.—Acute generalised ulcerative stomatitis, hyperacute congestion of abomasum and duodenum ; petechial hæmorrhages along ileum and large intestine ; cardiac petechial hæmorrhages ; congestion and œdema of both lungs ; tumor splenis.

Discussion.—It is apparent that *R. pinnatus* is a very poisonous plant for sheep, and produces when fed naturally very characteristic symptoms referable to lesions in the buccal cavity. Steyn[6] in his tests does not record any such symptoms possible because he relied on drenching. In both his cases a severe gastro-enteritis was produced, with diarrhœa, which was accompanied by great pain and

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prostration. Possibly the discrepancies between the symptoms and lesions described in the experimental cases from S. Africa and E. Africa may be largely due to the manner of administering the plants.

Plant No. 3.—Cucurbitaceæ. Adenopus abyssinucus H.K.F.

Vernacular names: Margi (Kikuyu); wild marrow.

Occurrence.—This genus, which according to Willis[7] contains twelve species, is found exclusively in Tropical Africa. The plant used in the present experiment grew luxuriantly in a much-used cattle paddock at Kabete on the slopes of a hill leading to a swamp, which was dry during the fine season. The species under investigation was recognised by its white diclinous flowers, which have a heavy, sickly perfume. The gourds grow to the size of a large orange, and are at first green, but as they ripen they take on a mottled appearance, there being large splashes of white on a green background. Section of the gourd shows a very thick mesocarp, which is white in colour and extremely bitter to taste.

Literature.—No reference to this plant ever having been tested toxicologically can be found in the available literature. As both species and genus are confined to the tropical regions of Africa, it is most likely that very little is known to European science about the plant. The Kikuyu regard the entire plant, but especially the gourd, as being highly poisonous.

Feeding Test.—Sheep No. 3155 received intravenously 15 c.c. of an aqueous solution of a ripe gourd, prepared by pouring 350 c.c. boiling water over 155 grams of finely chopped fruit. The infusion thus prepared was allowed to cool, after which it was filtered to remove all solid particles. Symptoms of illness appeared quickly after inoculation. Within three hours the animal had a worried look, salivated freely, and showed trembling of the jaws and lips. Respirations were increased, jerky, and accompanied by moaning. Diuresis was present, while there was all the symptoms of acute abdominal pain. By the seventh hour there was increased defæcation, the fæces having lost their normal pellet appearance and being "stool-like." The animal had a haggard, dull, anxious expression. After twenty-four hours the sheep was in the following condition: pupils were widely dilated, but vision was in no way impaired; it was very weak, listless, and unable to stand; diarrhœa was present, the excreta being soft, dark in colour, and contained much blood and mucous. Anorexia was complete, and diuresis was very marked. Just before death, which occurred about thirty-six hours after the inoculation of the plant infusion, diarrhœa became very profuse, pultaceous, and contained a lot of blood. The animal became progressively weaker and coma set in a short time before death.

Pathological Anatomy.—All the symptoms of hyperacute poisoning were present. Visible mucous membranes were deeply cyanosed, and blood-stained, frothy fluid escaped from the mouth and nostrils. Rigor mortis was very marked. Both pupils were widely dilated. Large and numerous petechial hæmorrhages were present on all the serous membranes, particularly on the endocardium

of the left ventricle. Both lungs were very deeply congested and œdematous, while the larynx, trachea and bronchii were filled with a pinkish whipped-up foam. The mucosa of the entire respiratory track was generally ecchymosed. There were twelve large hæmorrhages about the size of a sixpenny-piece in the pyloric portion of the abomasum, while the intestinal mucosa generally, large and small, showed numerous petechiæ. No ulcers were noticed anywhere, and the large bowel was quite empty of any contents. There was very marked stasis of dark-colour blood in the radicles of the mesenteric veins. All the parenchymatous organs were swollen and congested. The liver, however, in addition, was very soft, friable, and a light-putty colour.

Result of Test.—It will be seen that death occurred after inoculation of 15 c.c. infusion, which was equivalent to about 6.6 grams of the gourd. The rapidity of the course of the illness and the severity of the pathological lesions point to an extremely virulent poison. Unfortunately, there is no idea what the nature of this toxic substance is. It might well repay phamaceutical investigation, since the plant, being a relative of *Citrullus colocynthus Schrad*, from which the anthracine purgative colocynth is derived, might well yield some product to medical value. Anyhow, for the present it must be regarded as virulently poisonous for stock, and consequently should be eradicated from lands on which domestic cattle are grazed.

Plant No. 4.—Verbenaceæ. Lantana salvifolia.

Vernacular names : Songo (Jaluo) ; Muzireti (Kikuyu).

Occurrence.—This plant is widely distributed throughout Kenya, but is most prevalent in the Kikuyu district, where it grows luxuriantly on old shamba, or on lands which have been allowed to get out of control. It is fairly common in the country bordering Lake Victoria Nyanza, and is well known to the Baganda. The plant is a tall woody shrub, usually four to seven feet high, though taller specimens are not uncommon. It is readily recognised by its beautiful pinkish-mauve, salver-shaped flowers arranged in crowded spikes. Its fruits are said by the Kikuyu to be edible ; when ripe, they are purple in colour. Apparently the common Kenya species—*L. salvifolia*—is seldom touched by cattle, but goats eat the younger stems with evident relish. The Jaluo natives masticate the leaves, and use the salivary juice as an eye medicine. Apparently this concoction has a powerful astringent action, and is used indiscriminately for the treatment of ophthalmia and conjunctivitis in both man and beast. These natives regard the plant as poisonous if eaten in large amounts.

Literature.—This plant does not appear to have been investigated as regards its toxicological nature. Various workers in South Africa have not apparently experimented with this genus. In Australia, on the other hand, two species have been tested, *viz.*, *L. crocata* and *L. camara*. The former has been definitely proved poisonous by Seddon and Carne[8] in fairly large amounts (several pounds), though only 1½ lb. of the leaves produced symptoms of jaundice and mammary disturbances. *L. camara*, however, appears, according to the work of Seddon,

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Carne, and Mcgrath, to be innocuous when fed to stock in massive doses (80½ lb. and 396 lb. in two experiments). There was great difficulty in getting cattle to eat *L. camara*, as it possesses a very bitter taste. (Land cultivated by natives.)

Feeding Tests.—The first test ended fatally after 1,225 grams (2.7 lb.) had been given to a sheep. As death occurred suddenly and unexpectedly, a second experiment was carried out. This time 2,820 grams (6.2 lb.) failed to produce any outward symptoms. Possibly insufficient material was used, but even so, nearly three times as much as was originally used was given. The first sheep was fed during the last week of September, which is in the dry season, while the second animal was in experiment in December, during the short rains. Possibly the different results can be interpreted on the grounds that during the dry season the plant is poisonous, and during the rains innocuous. It is at present impossible to accept this hypothesis, and it is necessary to carry out further tests under various meteorological conditions to decide finally whether the plant is only poisonous at various seasons of the year or not at all. The sheep that died after eating 1,225 grams of the plant showed no symptoms of illness of any kind before death. It was found dead quite unexpectedly one morning, and as mentioned above, it was this fact that led to a further test.

Pathological Anatomy.—The most remarkable lesion was the presence of a large amount of straw-coloured fluid in all three serous cavities. In the pleural cavity alone it measured 300 c.c., and in the peritoneal and pericardial cavities 100 c.c. and 250 respectively. The mucosa of the upper respiratory and alimentary tracks was richly petechiated, while there was marked œdema and congestion of both lungs. A few ecchymoses were present on the endocardium of the left ventricle and along the intestinal tract.

Result of Test.—The pathological changes enumerated above show that the plant *Lantana salvifolia* possesses definite toxic properties. The nature of the active principle is quite unknown. The dropsical condition of all three serous cavities could not be associated with any other morbid condition as the sheep was quite healthy when put into experiment. The inference, therefore, is that this plant is poisonous for sheep, but that further tests should be conducted to ascertain whether it is only poisonous at a certain period of the year or throughout its life. This experiment serves to show that it is dangerous to dogmatise over the nature of a plant from a single experiment using one animal. If negative results had been obtained in the first instance the plant would have immediately been listed as non-poisonous, which would have apparently been gravely misleading.

Plant No. 5.—Umbelliferæ. Diplolophium abyssinicum, B. et H.F.

Vernacular names: Wild fennel; kirondet (Nandi); muraria (Kikuyu).

Occurrence.—Battiscombe[10] states that this herb is very common in Kenya on open grass lands between the altitudes of 6,000 to 8,000 feet. The whole plant resembles the cultivated fennel, and it has the characteristic odour. The

plant is readily recognised from this description and by its long tapering tap root. The Kikuyu declare that the plant is poisonous, but their evidence is not very convincing. On the other hand, the Naivasha Masai and the Nandi maintain that ingestion of the plant causes death amongst cattle and sheep. The material for the present experiment was sent to the laboratory by a farmer in the Rift Valley, near Lake Naivasha, with the history that it was responsible for poisoning some of his horses and cattle. No details were given, and only a little material, 75 grams, was received; and as, unfortunately, no further supply was forthcoming, drenching was relied upon, as feeding experiments were out of the question.

Literature.—This genus is exclusively found in tropical Africa, and contains only two genera. In the available literature no references can be found on the toxicological history of this plant, so that apparently it has not been tested previously.

Experiments with D. abyssinicum.—An infusion was made by pouring 150 c.c. boiling water on 75 grams of an entire plant finely minced. (*a*) Sheep 3099 received 30 c.c. of the infusion subcutaneously. Within an hour the sheep showed symptoms of great depression and nausea. It stood in a corner with its back arched and grinding its teeth. These symptoms, however, were very transient, and within six hours the animal was quite normal again. (*b*) Sheep 3042 received 10 c.c. of the infusion intra-jugularly. Immediately the animal lay down, and showed violent galloping movements of its limbs, and very marked twitching of the head. Diuresis was very marked; the respirations were increased to 150 per minute, and were of a " blowing " or bellows type. The animal ground its teeth very severely, and continually champed its jaws. There were all the signs of acute abdominal pain, but no diarrhœa was noted. These symptoms were very alarming for a couple of hours, and at one time it seemed as if death would supervene from failure of the respiratory system. However, all acute symptoms rapidly passed off, and within six hours the sheep was only drowsy and lethargic. For the following couple of days both animals were very much off colour, but there were no particular symptoms of ill-health.

Result.—It is impossible to state whether larger doses of this plant would have proved toxic for the sheep, but sufficiently alarming symptoms followed the intravenous and subcutaneous inoculation of amounts of plant which corresponded to 5 grams and 15 grams of whole plant. At present it is advisable to regard this herb as suspect of being poisonous, and to carry out further tests with larger quantities of material. By the picrate paper test the fresh plant was found to be free from hydrocyanic acid in recognisable amounts, so that the alarming symptoms which followed inoculation cannot be attributed to this agent.

Plant No. 6.—Umbelliferæ. Stephanorossia palustris, Chiev.

Vernacular names: Murerema (Kikuyu); chemkeruki (Lumbwa); water parsnip; water celery.

Occurrence.—This plant grows luxuriantly in damp, wet, or marshy places throughout the Colony, between the altitude of 6,000 to 10,000 feet. It is parti-

cularly common near rivers or streams, in which it may be totally or partially submerged during the wet season. Towards the end of the dry season, or at the end of a drought, when the smaller water-courses are dried up, the plant wilts and dies down. If, however, the dead vegetation on the beds of the dried-up water-courses is scraped away, the young plants can be seen throwing off young, fresh, green shoots, and trailing reddish-coloured hollow stems. As soon as the rains commence, and water once again starts to flow, the plant grows very rapidly, especially near the edges of the streams. When the herb grows near permanent water, as in the Nairobi district, and along the larger rivers in the Rift Valley, it is green throughout the year, and if the surrounding country is suffering from drought, this green vegetation is a severe temptation for cattle coming to water at rivers.

Incidence of Poisoning under Natural Conditions in Kenya.

The natives who herd in the alienated areas of Kenya, belonging to the Lumbwa, Nandi, Kikuyu and Masai tribes, believe that the plant is of a very poisonous nature. The writer has seen Masai in the Rift Valley chasing cattle away from the plant which grew in profusion along the Malawa River. Cases of poisoning by this plant usually occur towards the end of the dry season, before the onset of the long rains. It is at this time that all stock are driven by hunger to eat all sorts of plants that they would naturally avoid if there was plenty of grazing. No reference can be found in the literature that this plant has ever been previously experimentally tested. Cases of poisoning have been reported in Kenya from the Molo, Lumbwa, Naivasha, Kabete and Uasin Gishu districts. In the first place only was there a serious mortality, which involved thirty-two head of cattle. Even here it is impossible to say with any finality that the plant alone was responsible for all the deaths, since only a few of the dead cattle were actually post-mortemed. The farmer himself feared anthrax, and consequently lost no time in destroying the carcases. From the few bodies that were opened, numerous pieces of the plant in various degrees of maceration were found in the first three compartments of the gastro-intestinal canal. As all animals that died showed the same pre-mortal symptoms, there can be little doubt that all succumbed to the same cause. Every one of these beasts died near, or actually on the bed of, a dried-up stream in which the plant in a young sprouting stage was found. Careful survey of this locality showed that the cattle, in their eagerness for fresh food, had scraped away the dead vegetation on the surface of the dried-up stream bed, and literally rooted out the succulent young plants. Examination of the remaining vegetation, which consisted mainly of species of juncus, typha, pycreus and dracaena (all of which were subsequently proved non-poisonous for stock), showed that the cattle had browsed very heavily on all these plants. It is important to add that this particular part of the river had borne an evil reputation for stock for many years, and cattle were, under normal conditions, kept away from it. Other drinking pools, however, further along the river were regularly used, and here *S. palustris* was absent. A subsequent careful survey of that part

of the course of the stream which ran through the farm showed that this plant was only present at the place where the fatalities occurred. This discovery, in addition to finding pieces of the plant in the stomachs of a few of the dead cattle, strengthened the suspicion that the plant *S. palustris* was the cause of the mortality.

Experimental tests with material at various stages of growth were then commenced. As regards the cases of poisoning recorded from other districts which affected a few head at a time, only meagre details are available, as they were not investigated at first hand. In every instance, however, pieces of the plant were received from the carcases, and there was the history of sudden death. All cases occurred in the dry months of January and February, or the first few weeks of March. In a Naivasha case the beast (a bull) watered at a river at 2 p.m.; by 4 p.m. the animal was very ill and trembling violently, and at 6 p.m. it was dead. Those notes were supplied by the owner. Large quantities of *S. palustris*, which grew in profusion along the river at which the bull watered, were found in the stomach.

Experimental Tests with S. Palustris.—The material used for the first experiments was obtained from the locality at Molo, where the heavy mortality occurred. Besides *S. palustris*, other plants were collected for feeding tests, but all subsequently proved to be negative. The names of their genera have been noted above. At the time the experiments were undertaken all the plants were in the non-flowering stage, as the hot dry season was drawing to a close, and the long rains were already overdue. In the case of *S. palustris*, only the roots, stems and young sprouting leaves were obtained. At this time the identity of the plant was not known, and it was not until adult specimens were obtained that its genes and species were determined. Later, specimens were sent to Kew, where the identification made locally was confirmed.

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Experiments Nos. 1 *and* 2.—Sheep 2904 and sheep 3016 received 15 c.c. of a 10 per cent. aqueous infusion and 10 per cent. alcoholic solution respectively of *S. palustris* obtained from Molo. Both animals were found dead the following morning, about eighteen hours after inoculation. The following symptoms were recorded: Within ten minutes of inoculation remarkable symptoms began to manifest themselves. The animal became very dull and drowsy, and supported its head on various fixed objects. At times, however, both head and neck were sharply turned upwards, or to one side. About one hour after inoculation the sheep frequently rotated their heads, either in a complete circle or in a dorse-ventral direction while deflected to one side. Respirations were increased to 80 per minute, and were of an intermittent jerky nature. Very often there were five to fifteen rapid inspirations followed by a pause during which breathing stopped altogether. This type of respiration only lasted for one and a-half hours, and had entirely disappeared within two hours of inoculation. For the remainder of life breathing continued to be rapid, stertorous and jerky. Both sheep were dull and drowsy, and after three hours of illness showed onset of profuse fœtid diarrhœa. Prior to death a deep coma supervened, but the acute diarrhœa

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and nervous symptoms persisted. Details of the post-mortems are given later in the paper.

Experiments No. 3 and 4.—(*a*) With plant material collected in the young but non-flowering stage from the Muthari River, Nairobi district, feeding tests were carried out on sheep. A total of 2,405 grams of fresh green leaves and stems was fed without result. Unfortunately the supply of plant failed, as it was completely uprooted by a neighbouring agriculturist who was convinced of its toxicity. (*b*) Subsequently 75 c.c. of a tincture prepared from the same material was given intravenously to an ox. After inoculation the animal was very ill, and showed the following symptoms, *viz.*, staggering gait, dull stupid expression, and great difficulty in turning. It stood apart from its fellows, and cerebral symptoms were very marked, *e.g.*, inco-ordination, moving in circles, and giddiness. Those symptoms persisted for forty-eight hours, but gradually passed off. They were not unlike what was recorded on the sheep in experiment No. 1. Alcoholic intoxication can be ruled out of the question. There is no doubt if a larger dose of material had been given that death would have resulted.

Post-mortem Report on Sheep Nos. 2904 and 3016.

Acute congestion of stomachs and intestines, acute congestion and œdema of both lungs; petechiæ on endocardium and epicardium, congestion of liver, spleen, kidneys and myocardium.

Remarks.—It will be observed that plant material obtained in the young sprouting phase during the hot dry season was highly poisonous for two sheep, whereas later in the year, after the cessation of the long rains, the older non-flowering plant failed to produce symptoms of illness when fed to a sheep. Nevertheless, an alcoholic extract of the plant in the later phase caused very marked depression and narcosis in a bovine, which was so ill that at one time it was thought that death was imminent.

From these observations there can be no doubt that *S. palustris* is a highly poisonous plant, particularly so during the long, dry, hot season immediately preceding the rains. As far as present experiments show, the roots, fresh young stems, and first green leaves contain the toxic principle, no information beign available on the flowers and seeds. By the simple picrate paper test hydrocyanic acid was excluded from any rôle in the poisoning. Reference to the paper of Fleming and Peterson[11] shows that there are many points of similarity between *S. palustris* and *Cicuta occidentalis* from the toxicological point of view. For instance, " the leaves and stems might be eaten by cattle or sheep at certain periods of the year without harm, but the roots are always deadly " applies to both plants, and " the first green leaves and stems coming up from the ground are deadly, almost as much as the roots themselves." " The greatest danger comes from the tender green shoots which show up in ditches before the grass begins to grow, and may then be eaten greedily by animals hungry for green food." This last statement exactly describes the conditions which existed during

the serious poisoning at Molo. In the case of *C. occidentalis*, symptoms of poisoning appear in sheep sixteen to seventy-five minutes after a toxic dose of the plant, and in cattle after about one hour. It is superfluous to emphasise further great similarities between *S. palustris* and *C. occidentalis*, both known in their habitats in Kenya and Nevada respectively as " water parsnip." Both are very serious poisons from a stock owner's point of view, and every effort should be made to exterminate them from pastures over which stock feed. Like many other poisonous plants they take the greatest toll of life at the very season of the year when stock are most likely to browse on them. As they grow towards maturity their poisonous nature, if not entirely lost, is greatly reduced, but at this stage of their life history the plants are apparently rarely eaten, since after the rains grass is in abundance, and cattle only eat non-graminaceous herbage from perversity, or as the result of depraved appetite, non-acclimatisation, etc.

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Reviews

The Legal and Ethical Aspects of Medical Quackery. By L. M. Minty. Pp. xviii+262. London : Wm. Heinemann.

Veterinary surgeons sometimes complain that their province is invaded by quacks who, being subject to no disciplinary authority, can advertise and tout to obtain patients and make money out of the ignorant public. A perusal of this book would show any who were inclined to make such a complaint how infested the field of medical practice is with swarms of quacks, so that in comparison the veterinary surgeon would seem to have little to complain of. The author, it is true, says (p. 1) that though there is nothing to prevent an unqualified person practising surgery in this country, " as regards medicine, as distinct from surgery, the position is not quite clear." But at p. 5 he clearly states that " an unqualified person can still, notwithstanding anything in the Medical Acts, practise medicine and surgery without interference." Numerous instances of quackery are given, all of which are outside the Medical Acts. Chiropractists and osteopaths, herbalists, bonesetters, patent medicine vendors, and others of the tribe who batten on the credulity of the public, are shown up, and their methods of deceiving the public described. There is only a passing reference to Bullock's " Law Relating to Medical, Dental, and Veterinary

Practice," but it is obvious that the author has made considerable use of that work, as well as of several others not always fully acknowledged. His exposure of the methods of obtaining bogus medical diplomas from America prior to 1912 is illuminating, as is also his description of the co-called courses in chiropractic. The devious ways of the abortioner are described, and the wiles of the peripitetic optician explained. The chapter on Patent Medicine Quackery shows up the methods of obtaining testimonials, a great number of which are deliberate forgeries. The book concludes with a chapter on religious quackery, dealing especially with Lourdes and Christian Science.

Diseases and Ailments of Swine in Kenya Colony. By JAMES WALKER, O.B.E., M.R.C.V.S. *Chief Veterinary Research Officer, Colony and Protectorate of Kenya. Bulletin No.* 15. Department of Agriculture (Division of Veterinary Research), Colony and Protectorate of Kenya.

THE author, in *Bulletin No.* 15 of 1931, describes, for the information of breeders and owners, the commoner diseases and ailments of swine, including those known to occur in Kenya Colony, and methods for their diagnosis; and recommends treatment and measures for their control. By way of introduction, it is pointed out that swine diseases are comparatively rare in the colony, and are not a serious menace to the industry. The swine fever as known in Great Britain, Continental countries, and America, has so far not been observed; swine plague is unknown; tuberculosis is seldom met with; the rareness of this disease in cattle, and the consequent freedom of milk and milk products, which constitute one of the chief diets of swine, would explain the cause of its non-prevalence in the latter. Certain parasitic infections (Ascaris suis in particular) are of pathogenic and economic importance, but they can be controlled. A highly infectious disease, commonly known as East African swine fever, exists, but fortunately its distribution, as far as is at present known, is not widespread. The diseases and ailments which have been recognised in the colony, together with some of the commoner diseases experienced elsewhere, are grouped and described separately. Those occurring in Kenya include the following:

(1) *Filtrable Virus Diseases.*—(*a*) East African swine fever; (*b*) foot-and-mouth disease.

(2) *Bacillary Infections.*—(*a*) Anthrax; (*b*) bacillary necrosis; (*c*) tuberculosis (rare).

(3) *Protozoal Infections.*—(*a*) Spirochætosis.

(4) *Endoparasitic Infections.*—(*a*) Ascaris serum; (*b*) cysticercosis; (*c*) echinococcosis; (*d*) Arduenna strongyleria; (*e*) Physocephalus sexalatus; (*f*) œsophogostoma sp.; (*g*) trichuris sp.; (*h*) lung worm (metastrongylus).

(5) *Ectoparasites.*—(*a*) Sarcoptes scabies; (*b*) dermodectic mange; (*c*) tick infestation, in addition to the usual hæmatopinus series and pulex infestations.

(6) *Ailments* (*non-infectious*).—Rickets (rare); rheumatism (rare); sequelæ of castration (B. necrosis infection); cystic kidney; sun and heat stroke; indigestion; constipation; diarrhœa (scours).

East African Swine Fever.—The qualifying term "East African" was introduced to distinguish the East African disease from the British swine fever, which it resembles in many respects, but which appears to be ætiologically distinct. The nature and resistance of the virus, animals susceptible, transmission, incubation period, symptoms, post-mortem appearances, diagnosis, immunisation, and preventive measures, are briefly dealt with. Further work on this disease has since been carried out by the writer of the Bulletin, and will be recorded in the near future.

Personal

IN the New Year's Honours List we note that a knighthood has been conferred by H.M. The King on Professor F. T. G. Hobday, C.M.G., F.R.C.V.S., F.R.S.E., Principal and Dean of the Royal Veterinary College, and Editor of THE VETERINARY JOURNAL.

The O.B.E. (Civil Division) has been conferred on Mr. John Smith, M.R.C.V.S., D.V.H., Director of Animal Health and Acting Secretary for Agriculture, N. Rhodesia.

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